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JUNE, 1922

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Book Reviews.

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Archives of Neurology and Psychiatry

Vol. 7 JUNE, 1922 No. 6

AN ESSAY ON THE SHAKING PALSY, BY JAMES PARKINSON, M.D., MEMBER OF THE ROYAL COLLEGE OF SURGEONS *

WITH A BIBLIOGRAPHIC NOTE THEREON BY

ALFRED J. OSTHEIMER

A.B, M.D., M.R.C.S. (England)

L.R.C.P. (London)

Surgeon (R.), U. S. P. H. S.

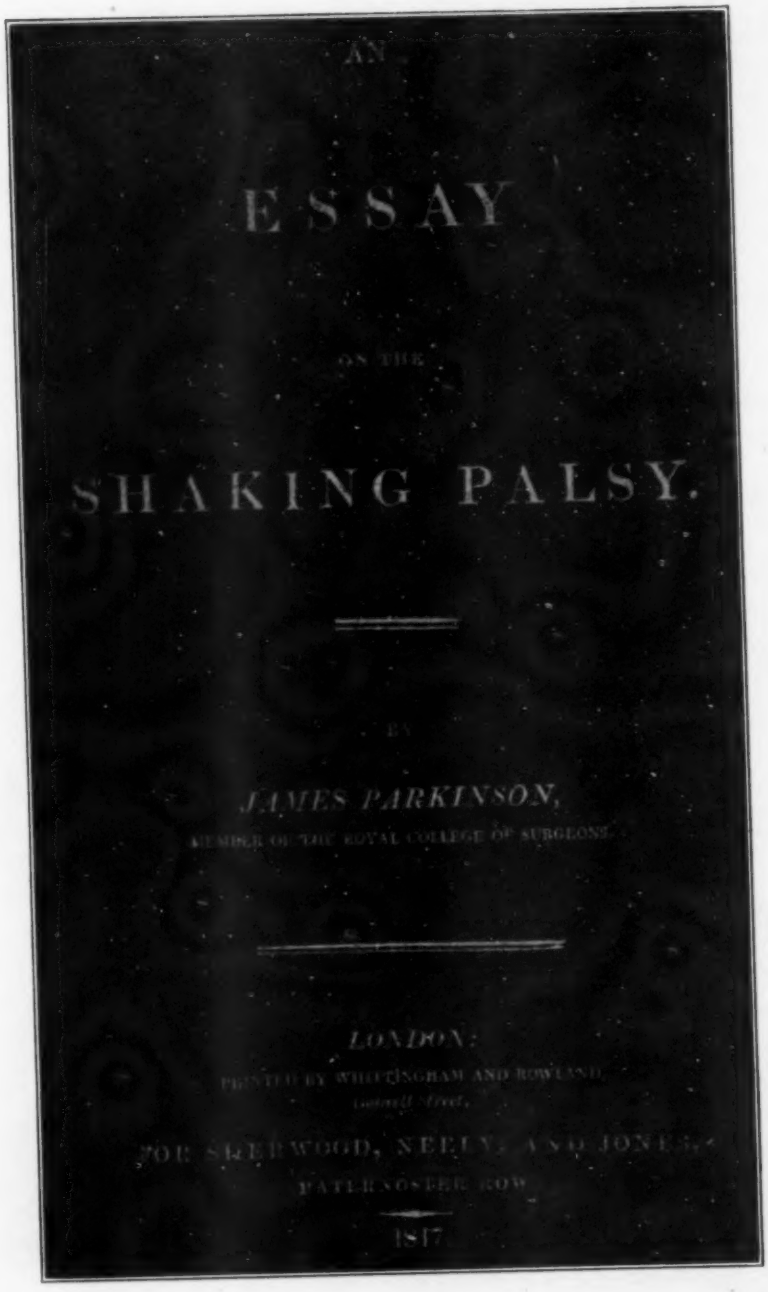
PHILADELPHIA

A good many years ago my interest in the history of this book was aroused by K. Mendel's statement in his monograph on paralysis agitans to the effect that, notwithstanding his thorough search through the libraries of the German universities, as well as through the Royal Library, and through the larger English libraries, he did not succeed in finding one copy of Parkinson's original description of the disease contained in an octavo booklet, entitled "An Essay on the Shaking Palsy," published in 1817 by Sherwood, Nelly & Jones, London.

Charcot in his twentieth "Leçon de Mardi," given on June 12, 1888, in his clinic at La Salpetrière, said that it was only after a long search that he was able, through the kindness of the librarian of the University of Manchester, to see and read a copy of this little book. On that morning he advised his students to translate the work into French on account of the excellent description of the disease contained therein.

Erich Ebstein, in a bibliographic note contained in the Feb. 15, 1912, number of the *Neurologisches Centralblatt,* calls attention to the fact that Charcot did not own a copy, as it was never found in his library, and that there was not a copy in the Paris Academy of Medicine, and none in the Bibliothèque Nationale. Ebstein further states that there are only four copies known to exist—one in the Surgeon-General's Library, Washington, D. C.; one in the Library of the Royal College of Physicians, Edinburgh; one in the Library of the Manchester Medical Society, Manchester, and one in the Library of the

* Photostat copy obtained from the Library of the Surgeon General, Washington, D. C.



AN

ESSAY

ON THE

SHAKING PALSY.

BY

JAMES PARKINSON,

MEMBER OF THE ROYAL COLLEGE OF SURGEONS.

LONDON:

PRINTED BY WHITTINGHAM AND ROWLAND,

Goswell Street,

FOR SHERWOOD, NEELY, AND JONES,

PATERNOSTER ROW.

1817

Royal College of Surgeons, London. (Thus it is seen that Mendel's search could not have been very thorough, notwithstanding his statement.)

I have accidentally discovered that there is at least one more copy in existence, namely, the one in the private library of Dr. Sydney Kuh of Chicago. Although not one of Charcot's students took his advice about translating the booklet, a German named Willige did, and Barth of Leipzig published the translation in 1912, of course in German, as a part of Sudhoff's "Medical Classics."

It has thus come about that while German medical students and practitioners have easy access to this classic work, American and English students have up to the present time never been able to enjoy the esthetic pleasure of, nor to obtain the instruction to be gleaned from, the exact words of the original describer of the disease.

Charcot calls attention to the excellence of Parkinson's description, which, he says, covers the great majority of the cases, and must, if for no other reason, always be considered as one of the best because it was the very first.

I would like to quote the first definition of the disease: "Shaking Palsy (*Paralysis Agitans*). Involuntary tremulous motion, with lessened muscular power, in parts not in action and even when supported; with a propensity to bend the trunk forwards, and to pass from a walking to a running pace; the senses and intellects being uninjured."

Now, more than 100 years after Parkinson's time, the opportunity has presented itself to reprint his original description, so that American and English students and practitioners may henceforth have easy access to this historic classic of medical literature.

AN

ESSAY

ON THE

SHAKING PALSY

BY

JAMES PARKINSON

PREFACE.

The advantages which have been derived from the caution with which hypothetical statements are admitted, are in no instance more obvious than in those sciences which more particularly belong to the healing art. It therefore is necessary, that some conciliatory explana-

tion should be offered for the present publication: in which, it is acknowledged, that mere conjecture takes the place of experiment; and, that analogy is the substitute for anatomical examination, the only sure foundation for pathological knowledge.

When, however, the nature of the subject, and the circumstances under which it has been here taken up, are considered, it is hoped that the offering of the following pages to the attention of the medical public, will not be severely censured. The disease, respecting which the present inquiry is made, is of a nature highly afflictive. Notwithstanding which, it has not yet obtained a place in the classification of nosologists; some have regarded its characteristic symptoms as distinct and different diseases, and others have given its name to diseases differing essentially from it; whilst the unhappy sufferer has considered it as an evil, from the domination of which he had no prospect of escape.

The disease is of long duration: to connect, therefore, the symptoms which occur in its later stages with those which mark its commencement, requires a continuance of observation of the same case, or at least a correct history of its symptoms, even for several years. Of both these advantages the writer has had the opportunities of availing himself; and has hence been led particularly to observe several other cases in which the disease existed in different stages of its progress. By these repeated observations, he hoped that he had been led to a probable conjecture as to the nature of the malady, and that analogy had suggested such means as might be productive of relief, and perhaps even of cure, if employed before the disease had been too long established. He therefore considered it to be a duty to submit his opinions to the examination of others, even in their present state of immaturity and imperfection.

To delay their publication did not, indeed, appear to be warrantable. The disease had escaped particular notice; and the task of ascertaining its nature and cause by anatomical investigation, did not seem likely to be taken up by those who, from their abilities and opportunities, were most likely to accomplish it. That these friends to humanity and medical science, who have already unveiled to us many of the morbid processes by which health and life is abridged, might be excited to extend their researches to this malady, was much desired; and it was hoped, that this might be procured by the publication of these remarks.

Should the necessary information be thus obtained, the writer will repine at no censure which the precipitate publication of mere conjectural suggestions may incur; but shall think himself fully rewarded by having excited the attention of those, who may point out the most appropriate means of relieving a tedious and most distressing malady.

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AN

ESSAY

ON THE

SHAKING PALSY

CHAPTER I.

DEFINITION—HISTORY—ILLUSTRATIVE CASES.

SHAKING PALSY. (*Paralysis Agitans.*)

Involuntary tremulous motion, with lessened muscular power, in parts not in action and even when supported; with a propensity to bend the trunk forwards, and to pass from a walking to a running pace: the senses and intellects being uninjured.

The term Shaking Palsy has been vaguely employed by medical writers in general. By some it has been used to designate ordinary cases of Palsy, in which some slight tremblings have occurred; whilst by others it has been applied to certain anomalous affections, not belonging to Palsy.

The shaking of the limbs belonging to this disease was particularly noticed, as will be seen when treating of the symptoms, by Galen, who marked its peculiar character by an appropriate term. The same symptom, it will also be seen, was accurately treated of by Sylvius de la Boe. Juncker also seems to have referred to this symptom: having divided tremor into active and passive, he says of the latter, "ad affectus semiparalyticos pertinent; de qualibus hic agimus, quique *tremores paralytoidei* vocantur." Tremor has been adopted, as a genus, by almost every nosologist; but always unmarked, in their several definitions, by such characters as would embrace this disease. The celebrated Cullen, with his accustomed accuracy observes, "Tremorem, utpote semper symptomaticum, in numerum generum recipere nollem; species autem a Sauvagesio recensitas, prout mihi vel astheniæ vel paralysios, vel convulsionis symptomata esse videntur, his subjungam.* Tremor can indeed only be considered as a symptom, although several species of it must be admitted. In the present instance, the agitation produced by the peculiar species of tremor, which here occurs, is chosen to furnish the epithet by which this species of Palsy, may be distinguished.

HISTORY.

So slight and nearly imperceptible are the first inroads of this malady, and so extremely slow is its progress, that it rarely happens, that the patient can form any recollection of the precise period of its commencement. The first symptoms perceived are, a slight sense of weakness, with a proneness to trembling in some particular part; sometimes in the head, but most commonly in one of the hands and arms. These symptoms gradually increase in the part first affected; and at an uncertain period, but seldom in less than twelvemonths or more, the morbid influence is felt in some other part. Thus assuming one of the hands and arms to be first attacked, the other at this period becomes similarly affected. After a few more months the patient is found to be less strict than usual in preserving an upright posture: this being most observable whilst walking, but sometimes whilst sitting or standing. Sometime after the appearance of this symptom, and during its slow increase, one of the legs is discovered slightly to tremble, and is also found to suffer fatigue sooner than the leg of the other side: and in a few months this limb becomes agitated by similar tremblings and suffers a similar loss of power.

Hitherto the patient will have experienced but little inconvenience; and befriended by the strong influence of habitual endurance, would

* Synopsis Nosologiæ Methodicæ.—Tom. ii. p. 195.

perhaps seldom think of his being the subject of disease, except when reminded of it by the unsteadiness of his hand, whilst writing or employing himself in any nicer kind of manipulation. But as the disease proceeds, similar employments are accomplished with considerable difficulty, the hand failing to answer with exactness to the dictates of the will. Walking becomes a task which cannot be performed without considerable attention. The legs are not raised to that height, or with that promptitude which the will directs, so that the utmost care is necessary to prevent frequent falls.

At this period the patient experiences much inconvenience, which unhappily is found daily to increase. The submission of the limbs to the directions of the will can hardly ever be obtained in the performance of the most ordinary offices of life. The fingers cannot be disposed of in the proposed directions, and applied with certainty to any proposed point. As time and the disease proceed, difficulties increase: writing can now be hardly at all accomplished; and reading, from the tremulous motion, is accomplished with some difficulty. Whilst at meals the fork not being duly directed frequently fails to raise the morsel from the plate: which, when seized, is with much difficulty conveyed to the mouth. At this period the patient seldom experiences a suspension of the agitation of his limbs. Commencing for instance in one arm, the wearisome agitation is borne until beyond sufferance, when by suddenly changing the posture it is for a time stopped in that limb, to commence, generally, in less than a minute in one of the legs, or in the arm of the other side. Harassed by this tormenting round, the patient has recourse to walking, a mode of exercise to which the sufferers from this malady are in general partial; owing to their attention being thereby somewhat diverted from their unpleasant feelings, by the care and exertion required to ensure its safe performance.

But as the malady proceeds, even this temporary mitigation of suffering from the agitation of the limbs is denied. The propensity to lean forward becomes invincible, and the patient is thereby forced to step on the toes and fore part of the feet, whilst the upper part of the body is thrown so far forward as to render it difficult to avoid falling on the face. In some cases, when this state of the malady is attained, the patient can no longer exercise himself by walking in his usual manner, but is thrown on the toes and forepart of the feet; being, at the same time, irresistibly impelled to take much quicker and shorter steps, and thereby to adopt unwillingly a running pace. In some cases it is found necessary entirely to substitute running for walking; since otherwise the patient, on proceeding only a very few paces, would inevitably fall.

In this stage, the sleep becomes much disturbed. The tremulous motion of the limbs occur during sleep, and augment until they awaken the patient, and frequently with much agitation and alarm. The power of conveying the food to the mouth is at length so much impeded that he is obliged to consent to be fed by others. The bowels, which had been all along torpid, now, in most cases, demand stimulating medicines of very considerable power: the expulsion of the fæces from the rectum sometimes requiring mechanical aid. As the disease proceeds towards its last stage, the trunk is almost permanently bowed, the muscular power is more decidedly diminished, and the tremulous agitation becomes violent. The patient walks now with great difficulty, and unable any longer to support himself with his stick, he dares not venture on this exercise, unless assisted by an attendant, who walking backwards before him, prevents his falling forwards, by the pressure of his hands against the fore part of his shoulders. His words are now scarcely intelligible; and he is not only no longer able to feed himself, but when the food is conveyed to the mouth, so much are the actions of the muscles of the tongue, pharynx, &c. impeded by impaired action and perpetual agitation, that the food is with difficulty retained in the mouth until masticated; and then as difficultly swallowed. Now also, from the same cause, another very unpleasant circumstance occurs: the saliva fails of being directed to the back part of the fauces, and hence is continually draining from the mouth, mixed with the particles of food, which he is no longer able to clear from the inside of the mouth.

As the debility increases and the influence of the will over the muscles fades away, the tremulous agitation becomes more vehement. It now seldom leaves him for a moment; but even when exhausted nature seizes a small portion of sleep, the motion becomes so violent as not only to shake the bed-hangings, but even the floor and sashes of the room. The chin is now almost immoveably bent down upon the sternum. The slops with which he is attempted to be fed, with the saliva, are continually trickling from the mouth. The power of articulation is lost. The urine and fæces are passed involuntarily; and at the last, constant sleepiness, with slight delirium, and other marks of extreme exhaustion, announce the wished-for release.

Case I.

Almost every circumstance noted in the preceding description, was observed in a case which occurred several years back, and which from the particular symptoms which manifested themselves in its progress; from the little knowledge of its nature, acknowledged to be possessed by the physician who attended; and from the mode of its termination; excited an eager wish to acquire some further knowledge of its nature and cause.

The subject of this case was a man rather more than fifty years of age, who had industriously followed the business of a gardener, leading a life of remarkable temperance and sobriety. The commencement of the malady was first manifested by a slight trembling of the left hand and arm, a circumstance which he was disposed to attribute to his having been engaged for several days in a kind of employment requiring considerable exertion of that limb. Although repeatedly questioned, he could recollect no other circumstance which he could consider as having been likely to have occasioned his malady. He had not suffered much from Rheumatism, or been subject to pains of the head, or had ever experienced any sudden seizure which could be referred to apoplexy or hemiplegia. In this case, every circumstance occurred which has been mentioned in the preceding history.

Case II.

The subject of the case which was next noticed was casually met with in the street. It was a man sixty-two years of age; the greater part of whose life had been spent as an attendant at a magistrate's office. He had suffered from the disease about eight or ten years. All the extremities were considerably agitated, the speech was very much interrupted, and the body much bowed and shaken. He walked almost entirely on the fore part of his feet, and would have fallen every step if he had not been supported by his stick. He described the disease as having come on very gradually, and as being, according to his full assurance, the consequence of considerable irregularities in his mode of living, and particularly of indulgence in spirituous liquors. He was the inmate of a poor-house of a distant parish, and being fully assured of the incurable nature of his complaint, declined making any attempts for relief.

Case III.

The next case was also noticed casually in the street. The subject of it was a man of about sixty-five years of age, of a remarkable athletic frame. The agitation of the limbs, and indeed of the head and of the whole body, was too vehement to allow it to be designated as trembling. He was entirely unable to walk; the body being so bowed, and the head thrown so forward, as to oblige him to go on a continued run, and to employ his stick every five or six steps to force him more into an upright posture, by projecting the point of it with great force against the pavement. He stated, that he had been a sailor, and attributed his complaints to having been for several months confined in a Spanish prison, where he had, during the whole period of his confinement, lain upon the bare damp earth. The disease had here continued so long, and made such a progress, as to afford little or no prospect of relief. He besides was a poor mendicant, requiring as well as the means of

medical experiment, those collateral aids which he could only obtain in an hospital. He was therefore recommended to make trial if any relief could, in that mode, be yielded him. The poor man, however, appeared to be by no means disposed to make the experiment.

Case IV.

The next case which presented itself was that of a gentleman about fifty-five years, who had first experienced the trembling of the arms about five years before. His application was on account of a considerable degree of inflammation over the lower ribs on the left side, which terminated in the formation of matter beneath the fascia. About a pint was removed on making the necessary opening; and a considerable quantity discharged daily for two or three weeks. On his recovery from this, no change appeared to have taken place in his original complaint; and the opportunity of learning its future progress was lost by his removal to a distant part of the country.

Case V.

In another case, the particulars of which could not be obtained, and the gentleman, the lamented subject of which was only seen at a distance, one of the characteristic symptoms of this malady, the inability for motion, except in a running pace, appeared to exist in an extraordinary degree. It seemed to be necessary that the gentleman should be supported by his attendant, standing before him with a hand placed on each shoulder, until, by gently swaying backward and forward, he had placed himself in equipoise; when, giving the word, he would start, in a running pace, the attendant sliding from before him and running forward, being ready to receive him and prevent his falling, after his having run about twenty paces.

Case VI.

In a case which presented itself to observation since those abovementioned, every information as to the progress of the malady was very readily obtained. The gentleman who was the subject of it is seventy-two years of age. He has led a life of temperance, and has never been exposed to any particular situation or circumstance which he can conceive likely to have occasioned, or disposed to this complaint; which he rather seems to regard as incidental upon his advanced age, than as an object of medical attention. He however recollects, that about twenty years ago, he was troubled with lumbago, which was severe and lasted some time. About eleven or twelve, or perhaps more, years ago, he first perceived weakness in the left hand and arm, and soon after found the trembling commence. In about three years afterwards the right arm became affected in a similar manner: and soon afterwards the convulsive motions affected the whole body, and began

to interrupt the speech. In about three years from that time the legs became affected. Of late years the action of the bowels had been very much retarded; and at two or three different periods had, with great difficulty, been made to yield to the action of very strong cathartics. But within the last twelvemonths this difficulty has not been so great; perhaps owing to an increased secretion of mucus, which envelopes the passing fæces, and which precedes and follows their discharge in considerable quantity.

About a year since, on waking in the night, he found that he had nearly lost the use of the right side, and that the face was much drawn to the left side. His medical attendant saw him the following day, when he found him languid, with a small and quick pulse, and without pain in the head or disposition to sleep. Nothing more therefore was done than to promote the action of the bowels, and apply a blister to the back of the neck, and in about a fortnight the limbs had entirely recovered from their palsied state. During the time of their having remained in this state, neither the arm nor the leg of the paralytic side was in the least affected with the tremulous agitation; but as their paralysed state was removed, the shaking returned.

At present he is almost constantly troubled with the agitation, which he describes as generally commencing in a slight degree, and gradually increasing, until it arises to such a height as to shake the room; when, by a sudden and somewhat violent change of posture, he is almost always able to stop it. But very soon afterwards it will commence in some other limb, in a small degree, and gradually increase in violence; but he does not remember the thus checking of it, to have been followed by an injurious effect. When the agitation had not been thus interrupted, he stated, that it gradually extended through all the limbs, and at last affected the whole trunk. To illustrate his observation as to the power of suspending the motion by a sudden change of posture, he, being then just come in from a walk, with every limb shaking, threw himself rather violently into a chair, and said, "Now I am as well as ever I was in my life." The shaking completely stopped; but returned within two minutes' time.

He now possessed but little power in giving a required direction to the motions of any part. He was scarcely able to feed himself. He had written hardly intelligibly for the last three years; and at present could not write at all. His attendants observed, that of late the trembling would sometimes begin in his sleep, and increase until it awakened him: when he always was in a state of agitation and alarm.

On being asked if he walked under much apprehension of falling forwards? he said he suffered much from it; and replied in the affirmative to the question, whether he experienced any difficulty in restraining himself from getting into a running pace? It being asked,

if whilst walking he felt much apprehension from the difficulty of raising his feet, if he saw a rising pebble in his path? he avowed, in a strong manner, his alarm on such occasions; and it was observed by his wife, that she believed, that in walking across the room, he would consider as a difficulty the having to step over a pin.

The preceding cases appear to belong to the same species: differing from each other, perhaps, only in the length of time which the disease had existed, and the stage at which it had arrived.

CHAP. II.

PATHOGNOMONIC SYMPTOMS EXAMINED—TREMOR COACTUS—SCELOTYRBE FESTINANS

It has been seen in the preceding history of the disease, and in the accompanying cases, that certain affections, the tremulous agitations, and the almost invincible propensity to run, when wishing only to walk, each of which has been considered by nosologists as distinct diseases, appear to be pathognomonic symptoms of this malady. To determine in which of these points of view these affections ought to be regarded, an examination into their nature, and an inquiry into the opinions of preceding writers respecting them, seem necessary to be attempted.

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1. *Involuntary tremulous motion, with lessened voluntary muscular power, in parts, not in action, and even supported.*

It is necessary that the peculiar nature of this tremulous motion should be ascertained, as well for the sake of giving to it its proper designation, as for assisting in forming probable conjectures, as to the nature of the malady, which it helps to characterise. Tremors were

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* Junckeri conspect. de tremore.

† Sect. v. Ubi auten solito pauciores deferunter ad eadem organa spiritus animales, imperfectæ ac imbecillæ observantur fieri eadem functiones, in motu tremulo et infirmo, nec diu durante, in visu debili, ac mox defatigato, &c.

Sect. xix. Inæqualiter, inordinatè, ac præter contraque volutatem moventur spiritus animales per nervos ad partes mobiles, in motu convulsivo, ac tremore, quassuve membtorum coacto.

Distinguendus namque his tremor quiescente licet ac decumbente corpore molustus a motu tremulo, de quo dictum. Sect. v. Quique quiescente corpore cessat, eodemque iterum moto repetit.

Sect. xxv. Coatus tremor debetur animalibus spiritibus inordinatè ac continuo, cum aliquo impetu ad trementium membrorum musculos per nervos propulsis: sive fuerit is universalis, sive particularis, sive corpus fuerit ad huc robustum sive debile, Sylvii de la Boë. Prax. lib. i. cap. xlii.

distinguished by Juncker into Active, those proceeding from sudden affection of the minds, as terror, anger, &c. and Passive, dependant on debilitating causes, such as advanced age, palsy, &c*. But a much more satisfactory and useful distinction is made by Sylvius de la Boë into those tremors which are produced by attempts at voluntary motion, and those which occur whilst the body is at rest†. Sauvages distinguishes the latter of these species (*Tremor Coactus*) by observing, that the tremulous parts leap, and as it were vibrate, even when supported: whilst every other tremor, he observes, ceases, when the voluntary exertion for moving the limb stops, or the part is supported, but returns when we will the limb to move; whence, he says, tremor is distinguished from every other kind of spasm*.

A small degree of attention will be sufficient to perceive, that Sauvages, by this just distinction, actually separates this kind of tremulous motion, and which is the kind peculiar to this disease, from the Genus Tremor. In doing this he is fully warranted by the observations of Galen on the same subject, as noticed by Van Swieten†. "Binas has tremoris species ‡ Galenus subtiliter distinxit, atque etiam diversis nominibus insignivit, tremor enim (τρόμος) facultatis corpus moventis et vehentis infirmitate oboritur. Quippe nemo, qui artus movere non instituerit tremet. Palpitantes autem partes, etiam in quiete fuerint, etiamsi nullum illis motum induxeris palpitant. Ideo primam (*posteriorem*) modo descriptam tremoris speciem, quando quiescenti homini involuntariis illis et alternis motibus agitantur membra, palpitationem (πάλμον) dixit, posteriorem (*primam*) vero, quæ non fit nisi homo conetur partes quasdam movere tremorem vocavit."

Under this authority the term palpitation may be employed to mark those morbid motions which chiefly characterise this disease, notwithstanding that this term has been anticipated by Sauvages, as characteristic of another species of tremor*. The separation of palpitation of

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* Nosolog. Methodic. Auctore Fr. Boissier de Sauvages, Tomi. II. Partis ii. p. 54, 1763.

† Comment. in Herman. Boerhaav. Aphorismos. Tom. ii. p. 181.

‡ De tremore. Cap. 3 and 4. Chart, Tom. vii. p. 200 201.

* Sect. xvi. *Tremor palpitans,* Preysinger classis morborum. *Palmos* Galeni. In tremoribus vulgaribus, æqualibus temporum intervallis, non musculus, sed artus ipsemet alternatim attollitur aut deprimitur, aut in oppositas partes it atque redit per minima tamen spatiola; in palpitatione verò sine ullo ordine musculi unius lacertus subito subsilit, nec regulariter continuoque movetur, sed nunc semel aut bis nunc minimè intra idem tempus subsilit; an causa irritans in sensorio communi, an in musculo ipse palpitante Quærenda sit, ignoramus. *Nosologiae Methodicae,* Vol. I. p. 559, 1768.

But the adoption which Sauvages has made of this term, will not be regarded as an absolute prohibition from the employment of it here; since the *tremor palpitans* of Sauvages should be considered rather as a palpitation of the muscles, whilst the motion which is so prominent a symptom in this disease, may be considered as a palpitation of the limbs.

the limbs (*Palmos* of Galen, *Tremor Coactus* of de la Boë) from tremor, is the more necessary to be insisted on, since the distinction may assist in leading to a knowledge of the seat of the disease. It is also necessary to bear in mind, that this affection is distinguishable from tremor, by the agitation, in the former, occurring whilst the affected part is supported and unemployed, and being even checked by the adoption of voluntary motion; whilst in the latter, the tremor is induced immediately on bringing the parts into action. Thus an artist, afflicted with the malady here treated of, whilst his hand and arm is palpitating strongly, will seize his pencil, and the motions will be suspended, allowing him to use it for a short period; but in tremor, if the hand be quite free from the affection, should the pen or pencil be taken up, the trembling immediately commences.

II. *A Propensity to Bend the Trunk Forwards, and to Pass from a Walking to a Running Pace.*—This affection, which observation seems to authorise the being considered as a symptom peculiar to this disease, has been mentioned by few nosologists: it appears to have been first noticed by Gaubius, who says, "Cases occur in which the muscles duly excited into action by the impulse of the will, do then, with an unbidden agility, and with an impetus not to be repressed, accelerate their motion, and run before the unwilling mind. It is a frequent fault of the muscles belonging to speech, nor yet of these alone: I have seen one, who was able to run, but not to walk*."

Sauvages, referring to this symptom, says, another disease which has been very rarely seen by authors, appears to be referable to the same genus (Scelotyrbe, of which he makes *Chorea sancti viti* the first species); which, he says, "I think cannot be more fitly named than hastening or hurrying Scelotyrbe (*Scelotyrbem festinantem, seu festiniam*)."

Scelotyrbe festinans, he says, is a peculiar species of scelotyrbe, in which the patients, whilst wishing to walk in the ordinary mode, are forced to run, which has been seen by Carguet and by the illustrious Gaubius; a similar affection of the speech, when the tongue thus outruns the mind, is termed volubility. Mons. de Sauvages attributes this complaint to a want of flexibility in the muscular fibres. Hence, he supposes, that the patients make shorter steps, and strive with a more than common exertion or impetus to overcome the resistance; walking with a quick and hastened step, as if hurried along against their will. *Chorea Viti,* he says, attacks the youth of both sexes, but this disease

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* Est et ubi musculi, recte quiden ad voluntatis nutum in actum concitati, injussa dein agilitate atque impetu non reprimendo motus suos accelerant, mentemque invitam præcurrunt. Vitium loquelæ suculis frequens, nec his solis tamen proprium? vidi enim, qui currere, non gradi, piterat *.

* Institution. Patholog. Medicinal. Auctore. H. D. Gaubio. 751.

only those advanced in years; and adds, that it has hitherto happened to him to have seen only two of these cases; and that he has nothing to offer respecting them, either in theory or practice*.

Having made the necessary inquiries respecting these two affections, *Tremor coactum* of Sylvius de la Boë and of Sauvages, and *Scelotyrbe festinans* of the latter nosologist, which appear to be characteristic symptoms of this disease, it becomes necessary, in the next place, to endeavour to distinguish this disease from others which may bear a resemblance to it in some particular respects.

CHAP. III.

SHAKING PALSY DISTINGUISHED FROM OTHER DISEASES WITH WHICH IT MAY BE CONFOUNDED

Treating of a disease resulting from an assemblage of symptoms, some of which do not appear to have yet engaged the general notice of the profession, particular care is required whilst endeavouring to mark its diagnostic characters. It is sufficient, in general, to point out the characteristic differences which are observable between diseases in some respects resembling each other. But in this case more is required: it is necessary to show that it is a disease which does not accord with any which are marked in the systematic arrangements of nosologists; and that the name by which it is here distinguished has been hitherto vaguely applied to diseases very different from each other, as well as from that to which it is now appropriated.

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* Ad idem genus morbi altera species rārissima ab auctoribus prætervisa referenda videtur, quam non aptius nominari posse putem quàm scelotyrbem festinantem, seu festiniam.

Sect. II. *Scelotyrbe festinans;* est peculiaris scelotyrbes species in qua ægri solito more dum gradi volunt currere coguntur, quod videre est apud D. Carquet, et observavit Leydæ illustr. Gaubius. *Patholog. instit.* 751, et in loquela hæc *volubilitas* dicitur quâ lingua præcurrit mentem. Video actu mulierem sexagenariam hoc affectam morbo siccitati nervorum tribuendo; laborat enim rheumatismo sicco, seu ab acrimonia sanguinis, dolores nocte a calore recrudescant, à thermis non sublevantur: ei præscripsi phlebotomiam, et præmissis jusculis ex lactucâ, et collo arietis, lene catharticum, inde vero lacticinia.

Est affinitas cum scelotyrbe, chorea viti, deest flexibilitas in fibris musculorum; unde motus breves edunt, et conatu seu impetu solito majori, cum resistentiam illam superare nituntur, velut inviti festinant, ac præcipiti seu concitato passu gradiutur. Chorea viti pueros, puellasve impuberes aggreditur; festinia vero senes, et duos tantum hactenus observare mihi contigit. Quam multos autem videmus morbos, paucissimosque observamus. De theoriâ et pràxi nihil habeo quod dicam; etenim sola experienta praxim cujusvis morbi determinat, et ex hac pro felici vel infausto successu theoria dein elicienda est. *Nosolog. Methodic.* Auctore, Fr. Boissier de Sauvages. Tomi. II. Part ii. p. 108.

Palsy, either consequent to compression of the brain, or dependent on partial exhaustion of the energy of that organ, may, when the palsied limbs become affected with tremulous motions, be confounded with this disease. In those cases the abolition or diminution of voluntary muscular action takes place suddenly, the sense of feeling being sometimes also impaired. But in this disease, the diminution of the influence of the will on the muscles comes on with extreme slowness, is always accompanied, and even preceded, by agitations of the affected parts, and never by a lessened sense of feeling. The dictates of the will are even, in the last stages of the disease, conveyed to the muscles; and the muscles act on this impulse, but their actions are perverted.

Anomalous cases of convulsive affections have been designated by the term Shaking Palsy: a term which appears to be improperly applied to these cases, independent of the want of accordance between them and that disease which has been here denominated Shaking Palsy. Dr. Kirkland, in his commentary on Apoplectic and Paralytic Affections, &c. cites the following case, related by Dr. Charlton, as belonging, he says, to the class of Shaking Palsies. "Mary Ford, of a sanguineous and robust constitution, had an involuntary motion of her right arm, occasioned by a fright, which first brought on convulsion fits, and most excruciating pain in the stomach, which vanished on a sudden, and her right arm was instantaneously flung into an involuntary and perpetual motion, like the swing of a pendulum, raising the hand, at every vibration higher than her head; but if by any means whatever it was stopped; the pain in her stomach came on again, and convulsion fits were the certain consequence, which went off when the vibration of her hand returned."

Another case, which the Doctor designates as "A Shaking Palsy," apparently from worms, he describes thus, "A poor boy, about twelve or thirteen years of age, was seized with a Shaking Palsy. His legs became useless, and together with his head and hands, were in continual agitation; after many weeks trial of various remedies, my assistance was desired.

"His bowels being cleared, I ordered him a grain of Opium a day in the gum pill; and in three or four days the shaking had nearly left him." By pursuing this plan, the medicine proving a vermifuge, he could soon walk, and was restored to perfect health.

Whether these cases should be classed under Shaking Palsy or not, is necessary to be here determined; since, if they are properly ranked, the cases which have been described in the preceding pages, differ so much from them as certainly to oppose their being classed together: and the disease, which is the subject of these pages, cannot be considered as the same with Shaking Palsy, as characterised by those cases.

The term Shaking Palsy is evidently inapplicable to the first of these cases, which appears to have belonged more properly to the genus *Convulsio,* of Cullen, or to *Hieranosos* of Linnæus and Vogel*.

The latter appears to be referable to that class of proteal forms of disease, generated by a disordered state of primæ viæ, sympathetically affecting the nervous influence in a distant part of the body.

Unless attention is paid to one circumstance, this disease will be confounded with those species of passive tremblings to which the term Shaking Palsies has frequently been applied. These are, *tremor temulentus,* the trembling consequent to indulgence in the drinking of spirituous liquors; that which proceeds from the immoderate employment of tea and coffee; that which appears to be dependent on advanced age; and all those tremblings which proceed from the various circumstances which induce a diminution of power in the nervous system. But by attending to that circumstance alone, which has been already noted as characteristic of mere tremor, the distinction will readily be made. If the trembling limb be supported, and none of its muscles be called into action, the trembling will cease. In the real Shaking Palsy the reverse of this takes place, the agitation continues in full force whilst the limb is at rest and unemployed; and even is sometimes diminished by calling the muscles into employment.

CHAP. IV

PROXIMATE CAUSE—REMOTE CAUSES—ILLUSTRATIVE CASES.

Before making the attempt to point out the nature and cause of this disease, it is necessary to plead, that it is made under very unfavorable circumstances. Unaided by previous inquiries immediately directed to this disease, and not having had the advantage, in a

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* Corporis agitatio continua, indolens, convulsiva, cum sensibilitate.—*Linn.*

Agitatio corporis vel artuum convulsiva continua, chronica, cum integritate sensuum.—*Vogel.*

This genus is resolved by Cullen into that of Convulsio. *Synops. Nosol.* 1803.

Dr. Macbride has given a very interesting and illustrative case of this disease.

"Hieranasós, or Morbus Sacer, so called, as being vulgarly supposed to arise from witchcraft, or some extraordinary celestial influence, is a distinct genus of disease, though a very uncommon one; the author once had an opportunity of seeing a case. The patient was a lad about seventeen, who at that time had laboured under this extraordinary disease for more than twelve years. His body was so distorted, and the legs and arms so twisted round it, by the continued convulsive working, that no words can give an adequate idea of the oddity of his figure; the agitation of the muscles was perpetual; but in general he did not complain of pain nor sickness; and had his senses perfectly; insomuch that he used to assist his mother, who kept a little school, in teaching children to read." *A methodical Introduction to the Theory and Practice of Physic. By David Macbride, M. D. p.* 559.

single case, of that light which anatomical examination yields, opinions and not facts can only be offered. Conjecture founded on analogy, and an attentive consideration of the peculiar symptoms of the disease, have been the only guides that could be obtained for this research, the result of which is, as it ought to be, offered with hesitation.

SUPPOSED PROXIMATE CAUSE.

A diseased state of the *medulla spinalis,* in that part which is contained in the canal, formed by the superior cervical vertebræ, and extending, as the disease proceeds, to the *medulla oblongata.*

By the nature of the symptoms we are taught, that the disease depends on some irregularity in the direction of the nervous influence; by the wide range of parts which are affected, that the injury is rather in the source of this influence than merely in the nerves of the parts; by the situation of the parts whose actions are impaired, and the order in which they become affected, that the proximate cause of the disease is in the superior part of the medulla spinalis; and by the absence of any injury to the senses and to the intellect, that the morbid state does not extend to the encephalon.

Uncertainty existing as to the nature of the proximate cause of this disease, its remote causes must necessarily be referred to with indecision. Assuming however the state just mentioned as the proximate cause, it may be concluded that this may be the result of injuries of the medulla itself, or of the theca helping to form the canal in which it is inclosed.

The great degree of mobility in that portion of the spine which is formed by the superior cervical vertebræ, must render it, and the contained parts, liable to injury from sudden distortions. Hence therefore may proceed inflammation of quicker or of slower progress, disease of the vertebræ, derangement of structure in the medulla, or in its membranes, thickening or even ulceration of the theca, effusion of fluids, &c.

But in no case which has been noticed, has the patient recollected receiving any injury of this kind, or any fixed pain in early life in these parts, which might have led to the opinion that the foundation for this malady had been thus laid. On the subject indeed of remote causes, no satisfactory accounts has yet been obtained from any of the sufferers. Whilst one has attributed this affliction to indulgence in spirituous liquors, and another to long lying on the damp ground; the others have been unable to suggest any circumstance whatever, which, in their opinion, could be considered as having given origin, or disposed, to the calamity under which they suffered.

Cases illustrative of the nature and cause of this malady are very rare. In the following case symptoms very similar are observable, so far as affecting the lower extremities. That the medulla spinalis

was here affected, and in its lower part, is not to be doubted: but this, unfortunately, was never ascertained by examination. It must be however remarked, that this case differed from those which have been given of this disease, in the suddenness with which the symptoms appeared.

A. B. aged twenty-six years, during a course of mercury for a venereal affection, was exposed to severely inclement weather, for several hours, and the next morning, complained of extreme pain in the back, and of total inability to employ voluntarily the muscles of the lower extremities, which were continually agitated with severe convulsive motions. The physician who attended him employed those means which seemed best calculated to relieve him; but with no beneficial effect. The lower extremities were perpetually agitated with strong palpitatory motions, and, frequently, three or four times in a minute, suddenly raised with great vehemence two or three feet from the ground, either in a forward or oblique direction, striking one limb against the other, or against the chairs, tables, or any substance which stood in the way. To check these inordinate motions, no means were in the least effectual, except striking the thighs forcibly during the more violent convulsions. No advantage was derived from all the means which were employed during upwards of twelvemonths. Full ten years after this period, the unhappy subject of this malady was casually met in the street, shifting himself along, seated in a chair; the convulsive motions having ceased, and the limbs having become totally inert, and insensible to any impulse of the will.

It must be acknowledged, that in the well-known cases, described by Mr. Potts, of that kind of Palsy of the lower limbs which is frequently found to accompany a curvature of the spine, and in which a carious state of the vertebræ is found to exist, no instructive analogy is discoverable; slight convulsive motions may indeed happen in the disease proceeding from curvature of the spine; but palpitating motions of the limbs, such as belong to the disease here described, do not appear to have been hitherto noticed.

Whilst striving to determine the nature and origin of this disease, it becomes necessary to give the following particulars of an interesting case of Palsy occasioned by a fall, attended with uncommon symptoms, related by Dr. Maty, in the third volume of the Medical Observations and Inquiries. The subject of this case, the Count de Lordat, had the misfortune to be overturned from a pretty high and steep bank. His head pitched against the top of the coach, and was bent from left to right; his left shoulder, arm, and especially his hand, were considerably bruised. At first he felt a good deal of pain along the left side of his neck, but neither then, nor at any other time, had he any faintings, vomitings, or giddiness.—On the sixth day he was let blood, on account

of the pain in his shoulder and the contusion of his hand, which were then the only symptoms he complained of, and of which he soon found himself relieved.—Towards the beginning of the following winter, he began to find *a small impediment in uttering some words, and his left arm appeared weaker.* In the following spring, having suffered considerably from the severities of the winter campaign, he found *the difficulty in speaking, and in moving his left arm, considerably increased.* —On employing the thermal waters of Bourbonne, his speech become freer, but, on his return to Paris, the Palsy was increased, and the arm somewhat wasted.—In the beginning of the next spring he went to Balaruc; when he became affected with *involuntary convulsive motions all over the body.* The left arm withered more and more, *a spitting began,* and now it was *with difficulty that he uttered a few words.* Frictions and sinapisms were successively tried, and an issue, made by a caustic, was kept open for some time without any effect; but no mention is made of what part the issue was established in.

Soon after this, and three years and a half after the fall, Doctor Maty first saw the patient, and gives the following description of his situation. "A more melancholy object I never beheld. The patient, naturally a handsome, middle-sized sanguine man, of a cheerful disposition, and an active mind, appeared much emaciated, stooping, and dejected. He *still walked alone with a cane, from one room to the other, but with great difficulty, and in a tottering manner;* his left hand and arm were much reduced, and would hardly perform any motion; *the right was somewhat benumbed, and he could scarcely lift it up to his head; his saliva was continually trickling out of his mouth, and he had neither the power of retaining it, nor of spitting it out freely.* What words he still could utter were monosyllables, and these came out, after much struggle, in a violent expiration, and with such a low voice and indistinct articulation, as hardly to be understood but by those who were constantly with him. He fetched his breath rather hard; his pulse was low, but neither accelerated nor intermitting. He took very little nourishment, could chew and swallow no solids, and even found great pain in getting down liquids. Milk was almost his only food; his body was rather loose, his urine natural, his sleep good, his senses, and the powers of his mind, unimpaired; he was attentive to, and sensible of every thing which was said in conversation, and shewed himself very desirous of joining in it; but was continually checked by the impediment in his speech, and the difficulty which his hearers were put to. Happily for him he was able to read, and as capable as ever of writing, as he shewed me, by putting into my hands an account of his present situation, drawn up by himself: and I am informed that he spent his time to the very last, in writing upon some of the most abstruse subjects."

This gentleman died about four years after the accident, when the body was examined by Dr. Bellett and Mons. Sorbier, who made the following report:

"We first examined the muscles of the tongue, which were found extenuated and of a loose texture. We observed no signs of compression in the lingual and brachial nerves, as high as their exit from the basis of the cranium and the vertebræ of the neck; but they appeared to us more compact than they commonly are, being nearly tendinous. The dura mater was in a sound state, but the pia mater was full of blood and lymph; on it several hydatids, and towards the falx some marks of suppuration were observed. The ventricles were filled with water, and the plexus choroides was considerably enlarged, and stuffed with grumous blood. The cortical surface of the brain appeared much browner than usual, but neither the medullary part nor cerebellum were impaired. We chiefly took notice of the Medulla Oblongata, this was greatly enlarged, surpassing the usual size by more than one third. It was likewise more compact. The membranes, which, in their continuation, inclose the spinal marrow, were so tough that we found great difficulty in cutting through them, and we observed this to be the cause of the tendinous texture of the cervical nerves. The marrow itself had acquired such solidity as to elude the pressure of our fingers, it resisted as a callous body, and could not be bruised. This hardness was observed all along the vertebræ of the neck, but lessened by degrees, and was not near so considerable in the vertebræ of the thorax. Though the patient was but nine and thirty years old, the cartilages of the sternum were ossified, and required as much labour to cut them asunder as the ribs; like these they were spungy, but somewhat whiter. The lungs and heart were sound. At the bottom of the stomach appeared an inflammation, which increased as it extended to the intestines. The ileum looked of that dark and livid hue, which is observed in membranous parts tending to mortification. The colon was not above an inch in diameter, the rectum was smaller still, but both appeared sound.—From these appearances, we were at no loss to fix the cause of this gradual palsy in the alteration of the medulla spinalis and oblongata."

Dr. Bellett offers the following explanation of these changes. "I conceive, that, by this accident, the head being violently bent to the right, the nervous membranes on the left were excessively stretched and irritated; that this cause extended by degrees to the spinal marrow, which being thereby compressed, brought on the paralytic symptoms, not only of the left arm, but at last in some measure also of the right. This induration seems to have been occasioned by the constant afflux of the nutritive juices, which were stopt at that place, and deprived of their most liquid parts; the grosser ones being unable to spread

in the boney cavity, by which they were confined, could only acquire a greater solidity, and change a soft body into a hard and nearly osseous mass. This likewise accounts for the increase of the medulla oblongata, which being loaded with more juices than it could send off, swelled in the same manner as the branches of trees, which will grow of a monstrous size, when the sap that runs into them is stopt in its progress. The medulla oblongata not growing so hard as the spinalis, was doubtless owing to its not being confined in an osseous theca, but surrounded with soft parts, which allowed it room to spread. The obstruction from the bulk of this substance must have affected the brain, and probably induced the thickening of the pia mater, the hydatids, and the beginning of suppuration, whereas the dura mater, being of a harder texture, was not injured*."

In some of the symptoms which appeared in this case, an agreement is observable between it and those cases which are mentioned in the beginning of these pages. The weakened state of both arms; the power first lessening in one arm, and then in a similar manner in the other arm; the affection of the speech; the difficulty in chewing and in swallowing; as well as of retaining, or freely discharging, the spittle; the convulsive motions of the body; and the unimpaired state of the intellects; constitute such a degree of accordance as, although it may not mark an identity of disease, serves at least to show that nearly the same parts were the seat of the disease in both instances. Thus we attain something like confirmation of the supposed proximate cause, and of one of the assumed occasional causes.

Whilst conjecturing as to the cause of this disease, the following collected observations on the effects of injury to the medulla spinalis, by Sir Everard Home, become particularly deserving of attention. It thence appears, that none of the characteristic symptoms of this malady are produced by compression, laceration, or complete division of the medulla spinalis.

"Pressure upon the medulla spinalis of the neck, by coagulated blood, produced paralytic affections of the arms and legs; all the functions of the internal organs were carried on for thirty-five days, but the urine and stools passed involuntarily *.

"Blood extravasated in the central part of the medulla, in the neck, was attended with paralytic affection of the legs, but not of the arms †.

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* Medical Observations and Inquiries, Vol. III. p. 257.

* A coagulum of blood, the thickness of a crownpiece, was found lying upon the external surface of the dura-matral covering of the medulla spinalis, extending from the fourth vertebra colli to the second vertebra dorsi. The medulla spinalis itself was uninjured.

† The sixth and seventh vertebra colli were dislocated, the medulla spinalis, externally, was uninjured; but in the centre of its substance, just at that part, there was a coagulum of blood nearly two inches in length.

"In a case where the substance of the medulla was lacerated in the neck, there was a paralysis in all the parts below the laceration, the lining of the œsophagus was so sensible, that solids could not be swallowed, on account of the pain they occasioned *.

"When the medulla of the back was completely divided, there was momentary loss of sight, loss of memory for fifteen minutes, and permanent insensibility in all the lower parts of the body. The skin above the division of the spinal marrow perspired, that below did not. The wounded spinal marrow appeared to be extremely sensible †". *Philosophical Transactions,* 1816, p. 485.

In two of the cases already noticed, symptoms of rheumatism had previously existed; and in Case IV. the right arm, in which the palpitation began, was said to have been very violently affected with rheumatic pain to the fingers ends. The consideration of this case, in which the palpitation had been preceded, at a considerable distance of time, by this painful affection of the arm, led to the supposition that this latter circumstance might be the cause of the palpitations, and the other subsequent symptoms of this disease. This supposition naturally occasioned the attention to be eagerly fixed on the following case; and of course influenced the mode of treatment which was adopted.

A. B. subject to rheumatic affection of the deltoid muscle, had felt the usual inconveniences from it for two or three days; but at night found the pain had extended down the arm, along the inside of the fore-arm, and on the sides of the fingers, in which a continual tingling was felt. The pain, without being extremely intense, was such as effectually to prevent sleep: and seemed to follow the course of the brachial nerve. Whilst ascertaining the propriety of this conclusion, the pain was found to ramify, as it were, on the fore and back part of the chest; and was slightly augmented by drawing a deep breath.

These circumstances suggested the probability of slight inflammation, or increased determination to the origin of the nerves of these parts, and to the neighbouring medulla. On this ground, blood was taken from the back part of the neck, by cupping; hot fomentations were applied for about the space of an hour, when the upper part of the back of the neck was covered with a blister, perspiration was freely induced by two or three small doses of antimonials, and the following morning the bowels were evacuated by an appropriate dose of calomel. On the following day the pains were much diminished, and in the course of four or five days were quite removed. The arm and hand felt now more than ordinarily heavy, and were evidently much

* The seventh vertebra colli was fractured, and the medulla spinalis passing through it, was lacerated and compressed.

† The spinal marrow, within the canal of the sixth vertebra dorsi, was completely destroyed by a musket ball. The person lived four days.

weakened: aching, and feeling extremely wearied after the least exertion. The strength of the arm was not completely recovered at the end of more than twelvemonths; and, after more than twice that time, exertion would excite the feeling of painful weariness, but no palpitation or other unpleasant symptom has occurred during the five or six years which have since passed.

The commencement, progress, and termination of this attack; with the success attending the mode of treatment, and the symptoms which followed, seem to lead to the conjecture, that the proximate cause of the disease, in this case, existed in the medulla spinalis, and that it might, if neglected, have gradually resolved itself into that disease which is the object of our present inquiry.

Some few months after the occurrence of the preceding case, the writer of these lines was called to a female about forty years of age, complaining of great pain in both the arms, extending from the shoulder to the finger ends. She stated, that she was attacked in the same manner as is described in the preceding case, about nine months before; that the complaint was considered as rheumatism, and was not benefited by any of the medicines which had been employed; but that after three or four weeks it gradually amended, leaving both the arms and hands in a very weakened and trembling state. From this state they were now somewhat recovered; but she was extremely anxious, fearing that if the present attack should not be soon checked, she might entirely lose the use of her hands and arms.

Instructed by the preceding case, similar means were here recommended. Leeches, stimulating fomentations, and a blister, which was made for sometime to yield a purulent discharge, were applied over the cervical vertebræ; and in the course of a very few days the pain was entirely removed. It is regretted that no farther information, as to the progress of this case, could be obtained.

On meeting with these two cases, it was thought that it might not be improbable that attacks of this kind, considered at the time merely as rheumatic affections, might lay the foundation of this lamentable disease, which might manifest itself at some distant period, when the circumstance in which it had originated, had, perhaps, almost escaped the memory. Indeed when it is considered that neither in the ordinary cases of Palsy of the lower extremities, proceeding from diseased spine, nor in cases of injured medulla from fractured vertebræ, any of the peculiar symptoms of this disease are observable, we necessarily doubt as to the probability of its being the direct effect of any sudden injury. But taking all circumstances into due consideration, particularly the very gradual manner in which the disease commences, and proceeds in its attacks; as well as the inability to ascribe its origin to any more obvious cause, we are led to seek for it in some slow morbid change

in the structure of the medulla, or its investing membranes, or theca occasioned by simple inflammation, or rheumatic or scrophulous affection.

It must be too obvious that the evidence adduced as to the nature of the proximate and occasional causes of this disease, is by no means conclusive. A reference to the test therefore which will be yielded by an examination of some of the more prominent symptoms, especially as to their agreement with the supposed proximate cause, is more particularly demanded. Satisfied as to the importance of this part of the present undertaking, no apology is offered for the extent to which the examination is carried on.

If the palpitation and the attendant weakness of the limbs, &c. be considered as to the order in which the several parts are attacked, it is believed, that some confirmation will be obtained of the opinion which has been just offered, respecting the cause, or at least the seat, of that change which may be considered as the proximate cause of this disease.

One of the arms, in all the cases which have been here mentioned, has been the part in which these symptoms have been first noticed; the legs, head, and trunk have then become gradually affected, and lastly, the muscles of the mouth and fauces have yielded to the morbid influence.

The arms, the parts first manifesting disorder action, of course direct us, whilst searching for the cause of these changes, to the branchial nerves. But finding the mischief extending to other parts, not supplied with these, but with the other nerves derived from nearly the same part of the medulla spinalis, we are of course led to consider that portion of the medulla spinalis itself, from which these nerves are derived, as the part in which those changes have taken place, which constitute the proximate cause of this disease.

From the subsequent affection of the lower extremities, and from the failure of power in the muscles of the trunk, such a change in the substance of the medulla spinalis may be inferred, as shall have considerably interrupted, and interfered with, the extension of the nervous influence to those parts, whose nerves are derived from any portion of the medulla below the part which has undergone the diseased change.

The difficulty in supporting the trunk erect, as well as the propensity to the adopting of a hurried pace, is also referable to such a diminution of the nervous power in the extensor muscles of the head and trunk, as prevents them from performing the offices of maintaining the head and body in an erect position.

From the impediment to speech, the difficulty in mastication and swallowing, the inability to retain, or freely to eject, the Saliva, may

with propriety be inferred an extension of the morbid change upwards through the medulla spinalis to the medulla oblongata, necessarily impairing the powers of the several nerves derived from that portion into which the morbid change may have reached. In the late occurrence of this set of symptoms, and the extension upwards of the diseased state, a very close agreement is observable between this disease and that which has been already shown, proved fatal to the Count de Lordat. But in this case, the disease doubtlessly became differently modified, and its symptoms considerably accelerated, in consequence of the magnitude of the injury by which the disease was induced.

CHAP. V.

CONSIDERATIONS RESPECTING THE MEANS OF CURE.

The inquiries made in the preceding pages yield, it is to be much regretted, but little more than evidence of inference: Nothing direct and satisfactory has been obtained. All that has been ventured to assume here, has been that the disease depends on a disordered state of that part of the medulla which is contained in the cervical vertebræ. But of what nature that morbid change is; and whether originating in the medulla itself, in its membranes, or in the containing theca, is, at present, the subject of doubt and conjecture. But although, at present, uniformed as to the precise nature of the disease, still it ought not to be considered as one against which there exists no countervailing remedy.

On the contrary, there appears to be sufficient reason for hoping that some remedial process may ere long be discovered, by which, at least, the progress of the disease may be stopped. It seldom happens that the agitation extends beyond the arms within the first two years; which period, therefore, if we were disposed to divide the disease into stages, might be said to comprise the first stage. In this period, it is very probable, that remedial means might be employed with success: and even, if unfortunately deferred to a later period, they might then arrest the farther progress of the disease, although the removing of the effects already produced, might be hardly to be expected.

From a review of the changes which had taken place in the case of Count de Lordat, it seems as if we were able to trace the order and mode in which the morbid changes may proceed in this disease. From any occasional cause, the thecal ligament, the membranes, or the medulla itself, may pass into the state of simple excitement or irritation, which may be gradually succeeded by such a local afflux and determination of blood into the minute vessels, as may terminate in actual but slow inflammation. The result of this would be a thickening of the theca, or membranes, and perhaps an increase in the volume of the medulla itself,

which would gradually occasion such a degree of pressure against the sides of the unyielding canal, as must eventually intercept the influence of the brain upon the inferior portion of the medullary column, and upon the parts on which the nerves of this portion are disposed.

From this review, and assuming that the morbid changes in this disease may not be widely dissimilar from those which occurred in the case of Count de Lordat, the chance of relief from the proposed mode of treatment may appear to be sufficient to warrant its trial.

In such a case then, at whatever period of the disease it might be proposed to attempt the cure, blood should be first taken from the upper part of the neck, unless contra-indicated by any particular circumstance. After which vesicatories should be applied to the same part, and a purulent discharge obtained by appropriate use of the Sabine Liniment; having recourse to the application of a fresh blister, when from the diminution of the discharging surface, pus is not secreted in a sufficient quantity. Should the blisters be found too inconvenient, or a sufficient quantity of discharge not be obtained thereby, an issue of at least an inch and a half in length might be established on each side of the vertebral columna, in its superior part. These, it is presumed, would be best formed with caustic, and kept open with any proper substance *.

Could it have been imagined that such considerable benefit: indeed, that such astonishing cures, could have been effected by issues in cases of Palsy of the lower extremities from diseased spine? although satisfied with ascribing those cases to scrofulous action, we are in fact as little informed respecting the nature of the affection, inducing the carious state of the vertebræ, as we are respecting the peculiar change of structure which takes place in this disease. Equally uninformed are we also as to the peculiar kind of morbid action, which takes place in the ligaments of the joints; as well as that which takes place in different instances of deep seated pains and affections of the parts contained in the head, thorax, and abdomen, and in all which cases the inducing of a purulent discharge in their neighborhood is so frequently productive of a cure. Experiment has not indeed been yet employed to prove, but analogy certainly warrants the hope, that similar advantages might be derived from the use of the means enumerated, in the present disease. It is obvious, that the chance of obtaining relief will depend in a great measure on the period at which the means are employed. As in every

* Cork, which has been hitherto neglected, appears to be very appropriate to this purpose. It possesses lightness, softness, elasticity and sufficient firmness; and is also capable of being readily fashioned to any convenient form. The form which it seems would be best adapted to the part, is that of an almond, or of the variety of bean called scarlet bean; but at least an inch and a half in length.

other disease, so here, the earlier the remedies are resorted to, the greater will be the probability of success. But in this disease there is one circumstance which demands particular attention; the long period to which it may be extended. One of its peculiar symptoms Scelotyrbe festinans, may not occur until the disease has existed ten or twelve years, or more; hence, when looking for the period, within which our hopes of remedial aid is to be limited; we may, guided by the slow progress of the malady, extend it to a great length, when compared with that within which we should be obliged to confine ourselves in most other diseases.

But it is much to be apprehended, as in many other cases, that the resolution of the patients will seldom be sufficient to enable them to persevere through the length of time which the proposed process will necessarily require. As slow as is the progress of the disease, so slow in all probability must be the period of the return to health. In most cases, especially in those in which the disease has been allowed to exist long unopposed, it may be found that all that art is capable of accomplishing, is that of checking its further progress. Nor will this be regarded as a trifle, when, by reference to the history of the disease, is seen the train of harassing evils which would be thus avoided.

But it seems as if there existed reason for hoping for more. For supposing change of structure to have taken place, it is extremely probable that this change may be merely increase in mass or volume by interstitial addition, the consequence of increased action in the minute vessels of the part. In that case, should the instituting of a purulent discharge, in a neighbouring part, act in the manner which we would presume it may—should it by keeping up a constant discharge, not merely alter the determination, but diminish the inordinate action of the vessels in the diseased part; and at the same time excite the absorbents to such increased action as may remove the added matter; there will exist strong ground for hope, that a happy, though slow restoration to health, may be obtained.

Until we are better informed respecting the nature of this disease, the employment of internal medicines is scarcely warrantable; unless analogy should point out some remedy the trial of which rational hope might authorize. Particular circumstances indeed must arise in different cases, in which the aid of medicine may be demanded: and the intelligent will never fail to avail themselves of any opportunity of making trial of the influence of mercury, which has in so many instances, manifested its power in correcting derangement of structure.

The weakened powers of the muscles in the affected parts is so prominent a symptom, as to be very liable to mislead the inattentive, who may regard the disease as a mere consequence of constitutional debility. If this notion be pursued, and tonic medicines, and highly

nutritious diet be directed, no benefit is likely to be thus obtained; since the disease depends not on general weakness, but merely on the interruption of the flow of the nervous influence to the affected parts.

It is indeed much to be regretted that this malady is generally regarded by the sufferers in this point of view, so discouraging to the employment of remedial means. Seldom occurring before the age of fifty, and frequently yielding but little inconvenience for several months, it is generally considered as the irremediable diminution of the nervous influence, naturally resulting from declining life; and remedies therefore are seldom sought for.

Although unable to trace the connection by which a disordered state of the stomach and bowels may induce a morbid action in a part of the medulla spinalis, yet taught by the instruction of Mr. Abernethy, little hesitation need be employed before we determine on the probability of such occurrence. The power, possessed by sympathy, in inducing such disordered action in a distant part, and the probability of such disordered action producing derangement of structure, can hardly be denied. The following Case seems to prove, at least, that the mysterious sympathetic influence which so closely simulates the forms of other diseases, may induce such symptoms as would seem to menace the formation of a disease not unlike to that which we have been here treating of.

A. B. A man, 54 years of age, of temperate habits and regular state of bowels, became gradually affected with slight numbness and prickling, with a feeling of weakness in both arms, accompanied by a sense of fulness about the shoulders, as if produced by the pressure of a strong ligature; and at times a slight trembling of the hands. During the night, the fulness, numbness, and prickling were much increased. The appetite had been diminished for several weeks; and the abdomen, on being examined, felt as though containing considerable accumulation.

Before adopting any other measures, and as there appeared to be no marks of vascular fulness, it was determined to empty the bowels. This was done effectually by moderate doses of calomel, with the occasional help of Epsom salts; and in about ten days, by these means alone, the complaints were entirely removed.

Before concluding these pages, it may be proper to observe once more, that an important object proposed to be obtained by them is, the leading of the attention of those who humanely employ anatomical examination in detecting the causes and nature of diseases, particularly to this malady. By their benevolent labours its real nature may be ascertained, and appropriate modes of relief, or even of cure, pointed out.

To such researches the healing art is already much indebted for the enlargement of its powers of lessening the evils of suffering humanity. Little is the public aware of the obligations it owes to those who, led by professional ardour, and the dictates of duty, have devoted themselves to these pursuits, under circumstances most unpleasant and forbidding. Every person of consideration and feeling, may judge of the advantages yielded by the philanthropic exertions of a HOWARD; but how few can estimate the benefits bestowed on mankind, by the labours of a MORGAGNI, HUNTER, or BAILLIE.

FINIS.

PARKINSON'S DISEASE

A CLINICAL STUDY OF ONE HUNDRED AND FORTY-SIX CASES *

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In this study only those cases were included in which the diagnosis was thought to be certain. Postencephalitic cases were not considered. For estimation of age and sex incidence 146 cases were used; for clinical studies, 140. The advantages of these records for source material as compared with hospital records are their uniformity, since they have all been under the direction of one person and written by a small number of historians (each has averaged about four years), and their brevity which, since only the more significant data are recorded, is in favor of greater accuracy. Their disadvantages are their occasional incompleteness and the limitation to positive data and to private cases. The restriction of statements to positive findings is compensated for by the fact that conclusions drawn from the recorded facts are minimal and therefore conservative. That age and sex distribution among private and dispensary cases runs along the same curve has been shown by Erb.[1] As numerous studies of the same subject have been previously recorded, only points of contrast and of special interest will be noted.

AGE DISTRIBUTION

By decades, the 146 cases fall into the usual sequence. Figure 1 shows the well established order of frequency. Of this group 80 per cent. are between 40 and 70 and 55 per cent. above 50 years of age at the onset of the disease. The age incidence reported by different authors is strikingly uniform. Figure 2 represents percentage distribution for the cases of Souques,[2] Dana,[3] and Patrick. The discrepancies appear only at the ends of the scale where cases are few and slight differences in number make comparatively large percentage differences.

When it is remembered that these curves are for groups of from 100 to 150 cases, each group representing a different part of Europe or America, their similarity is significant. It indicates that paralysis

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* Read before the Chicago Neurological Society. The cases are from the private records of one of us (H. T. P.).

1. Erb, Wilhelm, in Church: Diseases of the Nervous System, in Modern Clinical Medicine, 1908.

2. Souques, A.: Rev. neurol. **28**:552, 1921.

3. Dana, Charles L.: Nervous Diseases, New York, William Wood & Co., 1919.

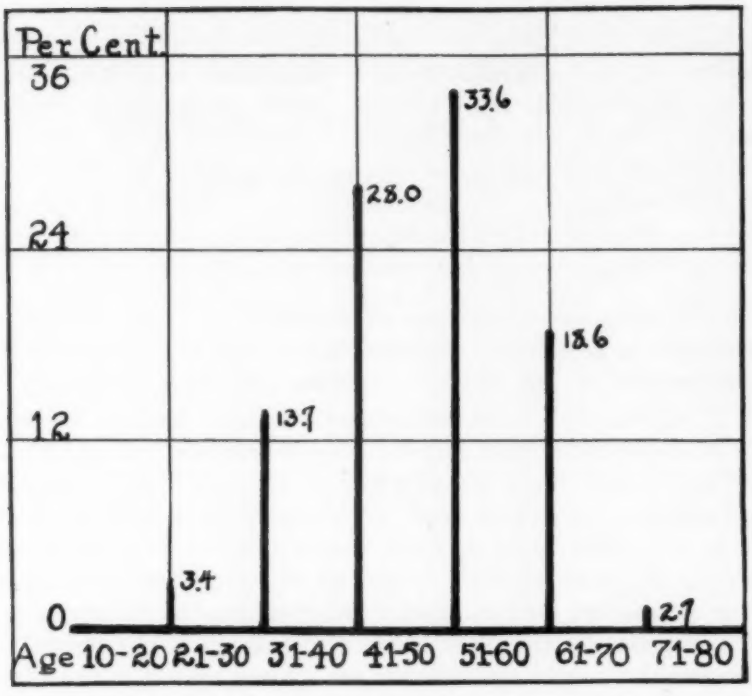

Fig. 1.—Percentage of distribution according to age at the onset of the disease in 146 cases of paralysis agitans.

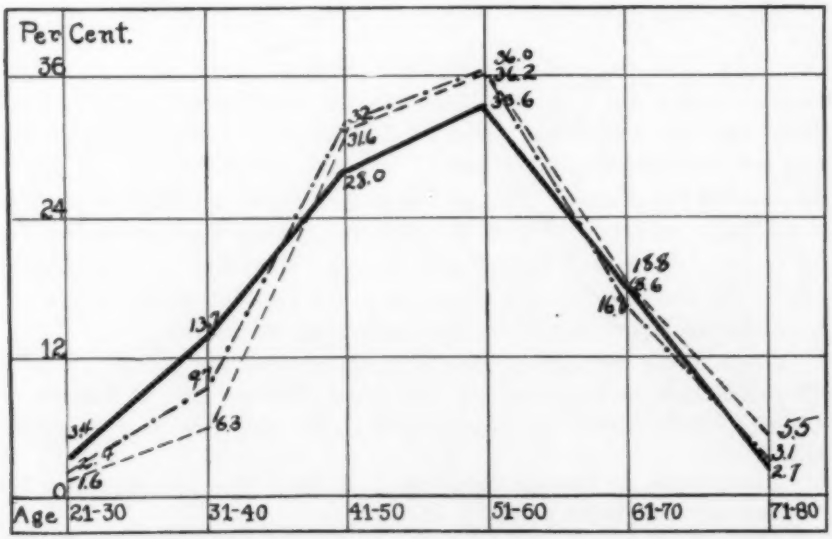

Fig. 2.—Percentage of distribution of paralysis agitans by age in decades. Souques' 125 cases are indicated by the dash and dot line, Dana's 127 cases by the dash line and Patrick's 146 cases by the continuous line.

agitans is a definite entity. Moreover, it adds weight to conclusions derived from the study of any one series, since they probably would hold for the others.

Age distribution in the sexes is practically the same. A small difference may be partly accounted for by the smaller number of cases in our female series (58), hence a greater element of chance (Figs. 3 and 4).

The age incidence of paralysis agitans taken regardless of the general population of each decade has limited value. It is obvious that one case in a group of ten shows relatively a much greater frequency value than five cases in a group of 100; and it follows that one case in a person in the seventies, a decade in which only 2 per cent. of the population are represented, is relatively more than three times as frequent as one case in a person in the fifties, a decade in which 7 per cent. of the

RELATIVE FREQUENCY VALUES

Age	No. of Cases*	Percentage Distribution by Decade, Native Population, 1910.	Frequency Values
20-30	5	17.7	1.8
31-40	20	13.1	10.5
41-50	41	9.2	30.7
51-60	49	6.9	49.0
61-70	27	4.4	42.3
71-80	4	2.1	13.1
	146		147.4

population are represented. Taking the number of cases in the fifties as a basis for comparison, the values in Figure 1 have been interpreted in terms of relative frequency values in the table. The percentage distribution of the native population by decades, on the basis of which frequency values are derived, is also shown in this table, which indicates that this disease is relatively more frequent in the sixties than in the forties, more frequent in the seventies than in the thirties, and rare in the twenties. It is relatively two and a half times as frequent in persons over 50 as in persons under 50. This is in favor of the theory that the disease is a degenerative process.

SEX

The ratio of males to females in this series is as 3 to 2. A preponderance of males over females has been found by most workers studying large series. Erb's ratio like that of Gowers [4] was 2 to 1 in

4. Gowers: Diseases of the Nervous System, 1893, Vol. 7, p. 637.

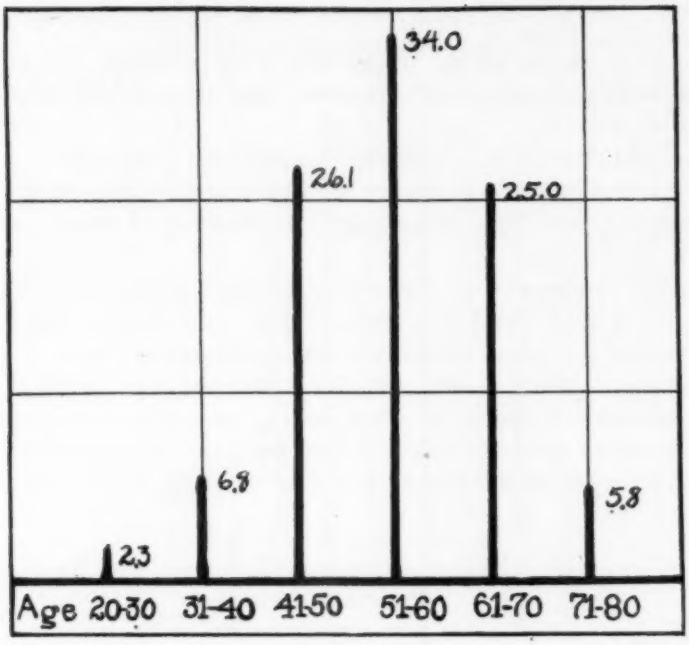

Fig. 3.—Percentages of distribution in males according to age (eighty-eight cases).

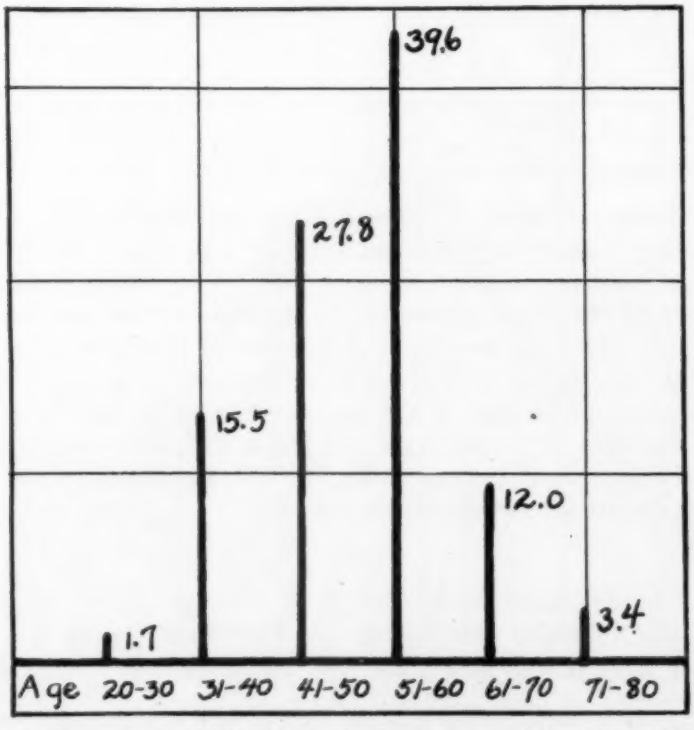

Fig. 4.—Percentage of distribution in females, according to age (fifty-eight cases).

favor of males. Reverse findings are largely confined to smaller series in which chance factors are greater.[5]

TRAUMA

There was a history of trauma in twenty-two cases. In three of these patients the trauma was operative—a left mastoid operation, removal of a cervical rib, and fracture of the jaw during extraction of a molar tooth. Apparently there is a high correlation between the site of trauma and the site of the first symptom of the disease, the tremor or rigidity taking its lead from the traumatized part, regardless of the time interval between the injury and the onset of the disease. For example, a man aged 39 at the onset of the disease, gave a history of a fall from a colt at 12 or 13 years of age, landing on his right shoulder and suffering from pain in that shoulder for a year. The right hand was first involved by the tremor. A man, aged 56 at the onset of the disease, which started in his left arm, had had his left hand cut off thirty-five years before. On the other hand, within two months following a fall in which the left arm was sprained, a man aged 65 at the onset of his disease, had the initial tremor in the left hand. Cases of paralysis agitans following trauma and affecting the injured side within a day have been described. In nine of our cases the trauma was of one designated extremity; in seven this extremity was the first involved; in one the opposite limb showed the first sign of the disease, and in one the other limb on the same side.

That the predilection may be for a part injured by disease is shown by the correlation of the onset with a part affected by rheumatic arthritis or some other nontraumatic disease. Of eight patients in whom a single extremity was thus affected, in six the first symptom of paralysis agitans appeared in the same extremity. Possibly not in agreement was the case of a man aged 48 at the time of the onset, who had had a right hemiplegia during the last ten years, and in whom the disease started on the unaffected side. As in the cases of trauma, there is no relation between the time of disease in an extremity and the time of onset of the initial symptom. In the nonoperative trauma group there were sixteen males and three females. The three operative cases were in females. The close relation between the area traumatized and the distribution of the symptoms has been especially emphasized by Mendel[5] and Oppenheim[6] and is apparently a frequent observation. Of course trauma is not to be considered as a direct etiologic factor,[7]

5. Mendel, K.: Die Paralysis Agitans, Berlin, S. Karger, 1911. See Mendel's review of these and other lists.

6. Oppenheim, H.: Text-Book of Nervous Diseases, Ed. 5 **2**:1298, 1911.

7. Pearce, F. S.: Trauma as an Exciting Cause of Paralysis Agitans, Phil. M. J., May 25, 1901, p. 1019.

but that it is a predisposing influence to the locus of onset of the disease, if it occurs, is borne out by these findings. Erb would add to the election of the traumatized part the factor of physical strain; he cites a case in which the part first affected in a painter was the left hand in which he held his palette.

To prove that trauma is an etiologic factor it would be necessary to show that the chances for getting the disease are greater in the traumatized part of the population. Such statistics are not at hand, nor does it seem likely that there is a less frequent history of trauma in the general population than in our lists (15 per cent.). This is no more significant than the predilection of rheumatic arthritis for a part receiving injury or bearing strain, or the predilection of syphilitic gummas for the frontal bones of Mohammedans, who in bowing to Allah repeatedly scrape their foreheads on cold marble.[8]

MENTAL SYMPTOMS

As described by most authors such symptoms are largely depressive reactions to the hopelessness of the disease. Wilson[9] writes that the mental symptoms are "limited to a depression of mood, the natural outcome perhaps of an incurable disease, and to an irritability also explicable by the nature of the ailment; anything beyond these is to be ascribed either to a concomitant arteriosclerosis or to some incidental condition." As recorded in this series, mental symptoms are naturally grouped as they occur before or after the disease and in the following order:

Group 1. History of acute mental symptoms following "shock" at or before onset of the first symptom of the disease. There are six cases in this group—three in males and three in females. In four of these the symptoms occurred within a year before the onset of paralysis agitans. Symptoms under this heading were: marked grief following death of wife and in this state when paralysis agitans began; frightened by burglar and in bed one month with weakness and "nerves"; breakdown and in a sanatorium for three months; great emotional shock and strain. The ages of the patients at the onset of acute mental symptoms were: 31, 46, 20, 51, 45 and 66; and at the onset of paralysis agitans in the same order: 31, 47, 50, 51, 53 and 66.

Group 2. History of depression preceding onset; seven cases: three in males and four in females. Under this heading such cases as the following were included: (1) Depressed, crying spells, suicidal thoughts, quitting work on account of depression, gradual improvement with return of symptoms two years ago with first symptom of paralysis agitans; (2) periodic depression one to two years preceding; (3)

8. Lacapére and Laurent: Paris méd. **8**:94, 1918.

9. Wilson, S. A. K.: Diseases of the Central Nervous System, Oxford Medicine, Vol. 6, Oxford, 1921.

depression four years preceding, attempted suicide by gas followed two days after the first attempt by the first symptom of paralysis agitans; (4) depression for ten years preceding onset with suicidal feelings.

Group 3. This is a less definite group. The patients were "very nervous," "highstrung," "worrisome," of a "nervous temperament," "easily upset" and "excited at least provocation." This group comprises twenty cases: thirteen in males and seven in females. In this group three patients showed fear—fear of something dreadful, a choking sensation with fear of death, fear of the shaking palsy. Three were patients with some form of apathy—apathetic, loss of interest; loss of ambition preceding the first symptom by six months; crying spells and careless of appearance. Generally these symptoms had preceded the paralysis agitans.

Mental symptoms during the course of the disease are recorded in twenty cases, but five of these are considered in the first group as occurring also before the onset. Of the fifteen remaining patients (seven males, eight females), depression or loss of interest is noted in ten (depressed only since onset of paralysis agitans, cries while examined; crying spells over condition for past nine months; depressed, careless of appearance and frequent crying past two months; nervous, depressed, insomnia, convinced is going to die, etc.). Worries and fears are noted in two cases; mental slowing-up, confusion, memory defects in three. In these three groups there are forty-eight cases, or 34 per cent. Even discounting Group 3 as being ambiguous, there remain twenty-eight, or 20 per cent., of whom definite mental symptoms are recorded. Especially noteworthy is the fact that mental symptoms are recorded more frequently as occurring before the onset of paralysis agitans than after, and as far as these records go, a depressive reaction is as frequent before the onset of the classical signs of the disease as after. There is no record of bonhomie as noted by Oppenheim [6] and only one record of periods of excitement. Actual mental deterioration is not mentioned in these records. The percentages found by other writers are in fair agreement.

Heredity findings are as in other reports: "Neuropathic" heredity in about 25 per cent. and direct heredity in six cases, of which two are questionable. Of these, the mother, the father, and a brother, had paralysis agitans. In the two questionable cases, the father also had a marked tremor, and the father had "the same tremor." Insanity (not described), one; melancholia, one; epilepsy, four; and migraine, three, are noted in antecedents.

Menopause or menstrual disturbance occurred in only ten cases, not a significant number for a group of fifty-eight women—of these, six had mental symptoms.

The complications are similar to the list in Mendel's monograph: tabes, one; hemiplegia, two; trifacial neuralgia, two; hyperthyroidism, one; diabetes, one. Two brothers of the patient with tabes, aged 70 when first seen, had general paresis. One of the cases complicated with hemiplegia was that of a printer, aged 65 at the time of onset, with tremor in the right hand which later involved the left. About fourteen months after the initial tremor he had a right hemiplegia with disappearance of the tremor on the right. With gradual recovery of the use of the right arm the tremor returned. Such a case was first described by Parkinson—one of his six cases.

Besides the unusual cases included under "complications," the following may be mentioned: hemiparalysis agitans, fifteen cases. These were cases usually seen early in the disease in which the tremor at the time of the examination was limited to one side, with however the typical gait, attitude and facies. In three of these cases the duration was one year or less; in eight, from one to two years; in three from two to three years. In one case the disease was of eight years' duration, and with minimal rigidity. In only four of these cases was the rigidity severe.

Unusual in this as contrasted with other series is the number (five) of patients with one-sided facial involvement.[10] In one of these cases the diagnosis was in doubt for a long time.[11] These cases of one-sided facial involvement occurred in the hemiparalysis agitans group and represent a fairly complete unilateral involvement including the face. No reference to such a series could be found in the literature.

Intention tremor was found in eighteen, or about 13 per cent. of all cases, indicating that the typical tremor is not invariable, a point already made with sufficient emphasis in the literature. Absence of tremor (paralysis agitans sine agitatione) is recorded in only five cases, in contrast with Erb's 20 per cent. The latter, however, included also cases with minimal tremor, a group not considered in this list. In these cases the duration was in order: seven months, two years, three years, five years and six years. Cases without rigidity (sine rigiditate) are thought to be the rarest of all types; not one was found in Erb's group of 183 cases. This condition was recorded in two patients in our series, one in a woman aged 46, who for one year and three months had shown the typical attitude of face and body. This was preceded by severe constant pain in the right arm which disappeared when the typical attitude began. The second case was that of a man, aged 51, who had had typical tremor of the left hand and arm for eighteen months with tremor in both legs for six months. Not the slightest rigidity was determined in either case.

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10. Patrick, Hugh T.: An Unusual Case of Paralysis Agitans, Arch. Neurol. & Psychiat., Proc. Chicago Neurol. Soc., this issue, p. 790.

11. Patrick, Hugh T.: J. Nerv. & Ment. Dis. **30**:469, 1903.

Three cases are classified in the "bulbar" group. One patient had slight difficulty in swallowing, one had choking spells at night and the third had marked difficulty in swallowing food. In these three cases the disease had made some progress before the "bulbar" symptoms commenced.

The focal distribution is represented in Figure 5. The extremities involved at the time of the last examination with the order of progress are indicated by numbers. For each type the number of cases seen is in the lower right hand corner in Roman numerals.

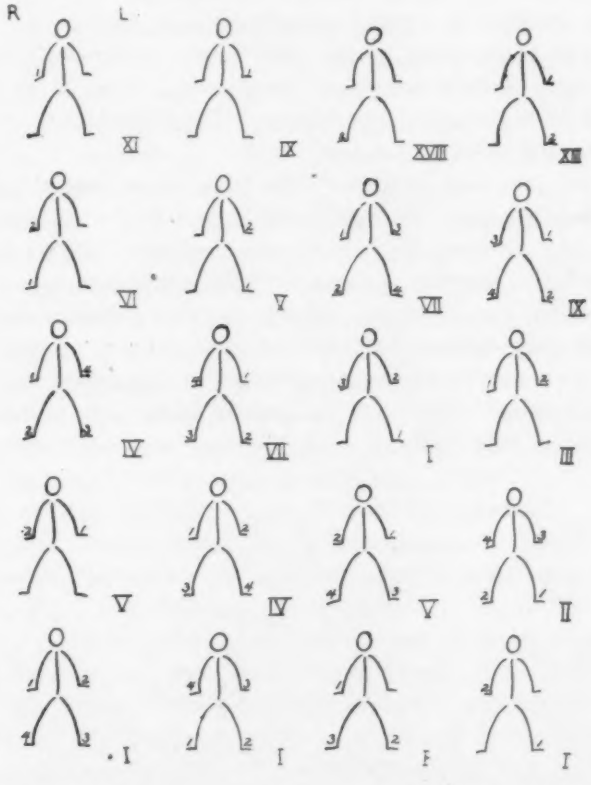

Fig. 5.—Focal distribution showing extremities involved at time of last examination with the order of progress are indicated by numbers. For each type the number of cases seen is in the lower right hand corner in Roman numerals.

SUMMARY

One hundred and forty-six private cases of "classical" paralysis agitans were used for studies in age and sex distribution; 140 for clinical studies. Frequency by decades was found to be greatest in the fifties and next in the forties; 80 per cent. of the patients were between 40 and 70 and 55 per cent. over 50 years of age. These find-

ings agree with other statistics. Considered in relation to the age incidence of the general population, the vast majority of patients are over 50 when the disease begins, and the onset is relatively more frequent in the seventies than in the thirties. The ratio of males to females was 3 to 2. Trauma occurred in twenty-two cases, and in these as in the histories of infected parts it was shown that the symptoms of paralysis agitans tend to start in a traumatized or diseased part. Since in this series there was a history of trauma in only 15 per cent. of all cases, it cannot be concluded that trauma is predisposing in any sense except as to the site of the initial symptom.

A large number of mental symptoms were shown by forty-eight patients, or in 34 per cent. of the cases. In contrast with the findings in other studies, mental symptoms were found to occur as frequently before as after the onset of the disease. These symptoms were largely in the form of depressive reaction.

Menstrual disturbances were found to be of no special significance. Heredity findings were not significant except in the form of "neuropathic heredity" in about 25 per cent. of the cases. Direct heredity of the disease was traced in six cases. The complications were: tabes, one; hemiplegia, two; trifacial neuralgia, two; hyperthyroidism, one, and diabetes, one. Fifteen patients had hemiparalysis agitans; eighteen cases were accompanied by intention tremor; five patients had one-sided facial involvement; four had paralysis agitans sine agitatione, two paralysis agitans sine rigiditate, and three had "bulbar symptoms."

HEREDITY IN EPILEPSY

A STUDY OF ONE THOUSAND FOUR HUNDRED AND FORTY-NINE CASES *

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There are still people who use "congenital" and "inherited" as synonyms, and who speak of inherited (meaning congenital) syphilis, forgetting that the parent is merely the carrier of a foreign body, a spirochete, to the fetus, and that there is nothing genetic in the process. Many accept the appearance of a disease or abnormality in successive generations as absolute proof of heredity: it is not, as is shown in "miner's phthisis," in which the exposure to the same environment is the cause, and not heredity. I shall use J. Arthur Thompson's definition of heredity. He says "by inheritance we mean all the qualities or characters which have their initial seat, their physical basis, in the fertilized egg cell; the expression of this inheritance in development results in the organism. Thus, heredity is no entity, no force, no principle, but a convenient term for the genetic relation between successive generations, and inheritance includes all that the organism is or has to start with in virtue of its hereditary relation."

We must also define "disease" and "a disease." T. Clifford Albutt defines disease most clearly and accurately as "a state of a living organism, a balance of function more unstable than that which we call 'health'; its causes may be imported, or the system may 'rock' from some implicit defect, but the disease itself is a perturbation which contains no elements essentially different from those of health, but elements presented in a different and less useful order." I shall use the phrase "a disease" to mean a perversion or cessation of functions or a function, having one specific cause, running a characteristic clinical course, acute or chronic, and showing either no anatomic changes or changes that are characteristic. Professor Martius gives this definition: "A disease is not an entity, not a character, but a process—an abnormal process injurious to the organism, which is set a-going by a *causa externa* and runs its course in some part of the body." Philosophically speaking, Martius' definition may be the more scientific, but in deciding, as we shall have to do, whether epilepsy is a disease, the one I use is the more useful for clinicians as it explains the elements which are necessary to bring a morbid process into the category of a

* Read before the Philadelphia Neurological Society, Jan. 27, 1922.

distinct disease. As I have defined the phrase, epilepsy is not a disease, but, after excluding all cases of epileptiform convulsions with known causes, there is a group left which we, for convenience sake, call idiopathic epilepsy; this paper is concerned only with that group. I have excluded all cases in which there was evidence of gross cerebral disease, such as injuries at birth, meningitis, fracture of the skull, brain injury and tumor. By gross cerebral disease, I mean disease visible to the naked eye. I presume that microscopic changes of one kind or another are always present, unless it be in the early months or years of the disease. Late changes indicate the seat of insult; they are not the cause of the primary symptoms.

It is possible that some time in the future epilepsy will cease to be regarded as a disease, just as dropsy and palsy ceased to be so regarded when their causes were discovered, but at the present time, though we know nothing of its cause and nothing definite of its morbid anatomy, it has such a characteristic symptomatology and clinical course that we are justified in still regarding it as an entity.

My data as to family morbidity are necessarily incomplete, because the members of any generation never have full knowledge of the medical history of the family, even of the parents, and either know nothing of the grandparents or only some startling and usually inaccurately remembered fact. Usually little is known about the infantile ailments of brothers and sisters, and if insanity or criminality is known to have occurred in the family, it is often denied. No study of heredity can be thorough unless accurate and complete biographies of at least three generations are known. When, in this paper, for example, I consider the diseases of the children of epileptic parents, conclusions must be formed with care because we do not know how many children there were, because many of the women are still well within the childbearing period, many others are too young to have had children, and not a few are unmarried and we know nothing of their childbearing history. Further, many of the children are far too young to exclude the possibility of epilepsy occurring later, and, of course, years too young to show the influence of parental disease in causing diseases which only appear in later life. Fortunately, however, the errors are of necessity those which minimize the influence of heredity. Thus, should all the children in this group live to middle life, let alone old age, and could we follow them, the number who would develop epilepsy, as well as other diseases, would be larger than is shown here.

It has been believed for centuries that the children of epileptic persons are more prone to epilepsy than others, but the scientific study of inheritance began only in recent times. Before taking up the examination of my own statistics I will quote a few opinions from literature. Gowers states that an inherited tendency (to epilepsy or

insanity) is traceable in 35 per cent. of the cases. He doubts whether any inherited morbid state outside the nervous system predisposes to epilepsy and specifically denies that phthisis does. He notes that idiopathic epilepsy sometimes occurs in subjects with inherited (he means congenital) syphilis when no other cause is traceable. Bernard Sachs and Leonard Guthrie speak of the close association of epilepsy and chorea in families, and Osler mentions it incidentally. William A. Turner writes: "The most common cause of epilepsy is ancestral epilepsy, but other neuropathic conditions, such as insanity and parental alcoholism, may be added. A careful inquiry into the parental history may give evidence of an hereditary neurosis, such as periodic headache, bilious attacks, neurasthenia, hysteria, or tics, while evidence of unsuspected or unrecognized epilepsy is often forthcoming." R. Gaupp regards "degeneration as the most important cause of genuine epilepsy." He lays stress on ancestral migraine and parental alcoholism as important predisposing causes.

EPILEPSY AND CONDITIONS CONDUCIVE TO EPILEPSY IN RELATIVES OTHER THAN OFFSPRING

Let us take up the statistical matter. The patients were all in attendance at the clinics of the Orthopaedic Hospital and Infirmary for Nervous Diseases. The histories of 442 of 1,891 patients were so fragmentary as to be valueless, leaving 1449 on which to work.

First as to the frequency of epilepsy in relatives other than offspring: The records show: father, 14; mother, 17; aunt, 14; uncle, 15; brother, 17; sister, 22; grandmother, 9; grandfather, 3; granduncle, 1; great grandmother, 1; first cousin, 13; making a total of 126 cases in which only one other member of the family than the patient was affected.

In addition there were the following instances of multiple occurrence in the family: aunt, cousin and brother, 1; grandfather and grandaunt, 1; uncle and aunt, 1; grandmother and aunt, 1; four maternal uncles, 1; two cousins (siblings), 1; mother's brother and niece, 1; father and cousin, 1; father, two brothers, three sisters, 1; paternal and maternal aunt, 1; maternal grandfather and paternal uncle, 1; twin brothers, 1; mother, brother, sister, 1; maternal and paternal first cousins, 1; two brothers and one sister, 1; maternal aunt and cousin, 2; uncle and cousin, 1; sister and cousin, 1; brother and maternal uncle, 1; maternal great grandmother, maternal grandmother, maternal aunt and sister, and patient's father had convulsions for a short period when 12 years old, 1; two of father's first cousins, 1; and both paternal grandparents, 1. To sum up: There were (excluding the children of epileptic persons) 193 relatives who are known to have had epilepsy. Of course, there were an unknown number of relatives who suffered

from the disease but whose illness was not known to our informants, and consequently the actual incidence was larger than our records show.

One hundred and sixty-five relatives other than offspring were known to have had infantile convulsions not followed by epilepsy. They are listed as follows: one sister, 31; one brother, 28; mother, 11; father, 6; paternal aunt, 1; paternal aunt and uncle, 1; maternal uncle, 2; paternal uncle, 1; two brothers and one sister, 1; one brother and one sister, 1; two siblings, 3; two brothers, 4; three brothers and one sister, 1; five siblings, 1; three siblings, 3; five brothers and one sister, 1; two sisters, 1; six siblings, 1; paternal uncle and two brothers, 1; cousin, 1; sister and brother, 4; one brother and four sisters, 1; eight siblings, 1; mother and uncle, 1; father and first cousin, 1; father, paternal aunt and uncle, 1. Ninety-one of the children died in or in consequence of convulsions. It was impossible to determine the causes of the convulsions in such a large number that the remainder was too small to use in drawing conclusions.

The relatives of thirty-six patients are recorded as insane, the distribution being as follows: father, 2; mother, 11; brother, 6; sister, 4; aunt, 4; uncle, 3; first cousin, 1; half-brother, 1; grandfather, 3; grandmother, 1. The following cases of multiple incidences occurred: two maternal uncles (father attempted suicide), 1; two first cousins, 1; aunt and grandaunt, 1; and grandmother insane and epileptic, 1. An inevitable error in the statistics of insanity is the fact that a family rarely realizes, and still more rarely admits, the existence of that disease until and unless the victim is sent to a hospital, and therefore the real percentage in my patients was higher than the figures indicate. Many people with hypomania or imbecility are believed by their relatives to be mentally well or, at worst, eccentric.

Cases of suicide and homicide are recorded as follows: One mother committed homicide; one father killed his father-in-law and himself; two mothers, five fathers, one sister and one of two brothers with melancholia committed suicide; and one father attempted it.

Chorea is recorded in relatives much more frequently than I expected would be the case. There were sixty-two cases recorded, classified thus: father, 3; mother, 7; sister, 13; brother, 9; uncle, 2; aunt, 4; children, 5; two brothers, 2; two sisters, 1; mother and sister, 2; father and sister, 1; father and brother, 1; mother and brother, 1; two aunts, 1; second cousin, 1. Tic was seldom mentioned, not, I think, because relatives call it chorea, but because the common habit spasm of children, and it is the most frequent type of tic, is not regarded seriously enough to be recalled years later. I am, nevertheless, convinced that tic is a serious sign of inherent instability.

DISEASE IN CHILDREN OF EPILEPTIC PERSONS

The records as to the occurrence of disease in the children of epileptic persons are very incomplete, and many of the women were still of childbearing age. (Of 128 married women under 40, 49 were between 19 and 29 years of age and 59 between 30 and 39.) The children already born varied in age from early infancy to middle life. In order to have accurate records it would be necessary to trace the history of the children until death. However, four children had had chorea, one also had epilepsy; six, epilepsy; and nine had had infantile convulsions. How many of them will become epileptic patients is impossible to foretell. The records as to imbecility are too fragmentary to be of any value.

The causes of death in the children of epileptic persons as recorded were about the same as in the general population. There were 123 deaths in infancy, 20 miscarriages, 18 stillbirths and 3 craniotomies. In ninety cases the cause of death was unknown. There were 443 children living, of whom 278 had epileptic fathers and 205 epileptic mothers. No conclusions can be drawn either from the number of children who lived or from the number who died, except that apparently a child with an epileptic father has a somewhat better chance to survive infancy than one with an epileptic mother.

Alcoholism and Epilepsy.—Much has been written about the relationship between parental alcoholism and epilepsy. My list records 155 fathers who were drunkards, eight cases in which both parents were drunkards, but only two in which the mother alone was a heavy drinker. Lists similar to this have often been used to prove that alcoholism alone, in the absence of all other causes, in the parent causes epilepsy in the offspring. The conclusion is, however, a little too hastily drawn. Students who approach the study of alcoholism from a philosophic point of view know that there are drunkards and drunkards. A craving for alcohol, and the same is true of other drugs, may in itself be a sign of degeneracy. The boy who during adolescence begins to drink and does so because he gets sensations from the drug a normal boy does not get, is inherently a different person than the middle aged man who, having retired from business and lacking intelligence enough to find occupation, sits about and drinks and continues to drink until he gets cirrhosis of the liver. The periodic drinker, who in many ways resembles the psychic epileptic, and who often has great mental ability, belongs in still another category. The man, and there are such, who can drink to drunkenness, sleep it off and be clear minded and hard working next day and continue this for years is still another type. My statistics, and all others I have read concerning alcoholism, fail to make any distinction, and for a very good reason: It is impossible in hospital cases to make a thorough study of the relatives of the patient, and

hence the records simply state whether or not the parents drink. Further, the personal equation of the examiner influences the record. What I would record as moderate drinking another might consider to be extreme and incurable alcoholism. The widely varying conclusions drawn by different writers using statistical material, in studying the question of alcoholism, is a classical example of proving anything from figures. The truth probably is that parental alcoholism is not the cause of epilepsy in the offspring, but is frequently a symptom of an inherent abnormality in the family, which in the parent shows itself in one way, in the offspring in another. If parental alcoholism were the cause of epilepsy, it would be a much more common disease. Whether heredity plays a large or small part in the production of the common drunkard, certainly the resemblance of the clinical picture in the periodic drinker, who at regularly or irregularly recurring intervals changes in personality, to the epileptic with his recurring fits, is not accidental; it means that both have an unstable organization, which surely is often inherited. I am not sure, even, that alcoholism is any more frequent in the families of epileptic persons than in other families. There are not, and there cannot be, any accurate statistics regarding the matter.

Phthisis and Epilepsy.—Whether there is any causal relation between parental or family phthisis and epilepsy in children is a difficult problem to solve. In my list 55 fathers, 31 mothers, 1 brother in eleven cases and 1 sister in nine, had phthisis. There were two cases in which 2 brothers, four cases in which 2 sisters, and one case in which 3 sisters were affected. In three cases both parents were tuberculous and in one case 4 siblings. There were thirty-four cases in which more than one of the immediate family had phthisis. This does not seem to be a larger percentage than is found in the general population. It is true that many more of the parents of the epileptic persons here studied had phthisis than had epilepsy, so that if mere occurrence in two generations proved heredity, phthisis would be a much greater factor than epilepsy itself. But epilepsy is only one of the nervous disorders which lay the ground for epilepsy or give the danger signal that it may occur. Insanity, certain types of alcoholism, chorea, all must be added together in summing up hereditary influence. Phthisis is a disease in kind unlike all of these. It cannot be added to them. That there is an inherited predisposition to it and also a natural immunity is undoubted, but that predisposition to disease of the lungs, in the parent, can, in the child, be transformed to a predisposition to epilepsy, is far from proved.

Frequency of Death of Children of Epileptic Parents.—I have made a list of cases in which more than one sibling of an epileptic parent died in infancy, because too frequent deaths among children in families already having a disease in the family, points toward (it does not prove)

some weaknesses in one or both parents, inherent or acquired. Of course, infectious infantile diseases may affect any family, barring the naturally immune, but if in any one family in which there exists a disease, for example, phthisis or epilepsy, death from other causes than infectious fevers is frequent in the young, it is some evidence of a general weakness. There were 46 cases in which 2 siblings died in infancy, 29 in which 3 died, 26 in which 4 died, 18 in which 5 died, 9 in which 6 died, 3 in which 7 died, 3 in which 8 died and 1 in which 9 died. It was impossible to get accurate statistics as to stillbirths and miscarriages. Any conclusion drawn from the number of deaths occurring in offspring would be a guess because we have no statistics of the incidence of deaths of children in families in the general population. My impression is that the juvenile death rate in my epileptic families is high.

I have been impressed by the infrequency with which migraine is mentioned. It is common, one of the commenest of diseases, and yet so rarely is it mentioned that I have not recorded the number of cases in which it was given as a symptom, because any conclusion drawn would be fallacious. The probable explanation is that it is such a common ailment people forget all about it.

COMMENT

Can we, from this long catalogue of terrible abnormalities (621 cases of serious nervous and mental disease) occurring in relatives of epileptic persons, a catalogue that is duplicated in the case histories of every hospital, draw any conclusion as to heredity in epilepsy? My statistics do not show that direct inheritance is important, since only thirty-four parents were known to be affected. On the other hand, the frequency of insanity, crime, chorea, alcoholism and epilepsy in relatives points toward congenital instability resulting from abnormality in the germ cell or sperm cell. Considering all the data, it is safe to assume that the effect of heredity is rarely direct, that usually it is indirect and general, not specific. In other words, a predisposition to nervous or mental disease is inherited; the resulting specific disease depends on external causes—it is environmental in the broadest meaning of the word.

What organ or organs are hereditarily at fault in epilepsy? One theory is that the motor cells in the brain are inherently overirritable and hence react violently to stimuli to which normal cells would be indifferent. If epilepsy consisted only in a succession of convulsions, this explanation would suffice. But the convulsion is only one symptom of the disease. Therefore the area of the brain affected must be wider than the motor cortex. Another theory is that because of disorders in the chemistry of the body, the whole brain is more or less

affected and because antidotes are also formed the convulsion occurs only when the poison overwhelms the antidote. We do not know what the disorder in metabolism is and what organs are directly involved. Some writers assume that the ductless glands, of which we are hearing so much and being taught something, are primarily at fault. This opinion is based on hypothesis. The most recent hypothesis, that the convulsions are psychogenic in origin and are a defense reaction of the subsconscious mind against an unpleasant situation, needs much more evidence than has been brought to its support. I suspect that biologic chemists rather than psychiatrists will solve the riddle.

INTRAVENOUS ADMINISTRATION OF IODIDS*

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Early in 1919, Weed and McKibben [1] presented the results of investigations which they had made on animals as to the value of hypertonic solutions of sodium chlorid in relation to cerebrospinal fluid pressure. They first observed an increase of salt in the spinal fluid.

With the various solutions the following observations were noted: A hypotonic solution (distilled water) caused an immediate rise in cerebrospinal fluid pressure. Ringer's solution (sodium chlorid, 0.9 per cent.; potassium chlorid 0.042 per cent.; calcium chlorid, 0.025 per cent.) caused an immediate rise but a fall again to normal within eighty minutes. Hypertonic sodium chlorid solutions caused an immediate rise and then a fall to below zero with a slow and gradual return to normal. An 18 per cent. sodium bicarbonate solution caused a fall to zero with a very slow return to normal. A 30 per cent. sodium sulphate solution manifested a slight rise, then a fall to below 50 mm. with a slow and gradual return to normal.

At the same time they observed a distinct alteration in the bulk of the brain by using hypertonic solutions of sodium chlorid. Their experiments were made on animals with the skull both opened and unopened. The brain was seen to fall away several millimeters from the inner surface of the skull after each injection.

During the past four years I have observed three cases of systemic fungus disease (oidiomycosis); in two of the three cases the budding oidium was obtained from the spinal fluid, and the patients were treated with iodids. The first case [2] could be kept under control only with very large doses of potassium iodid (by mouth), and in due time it was necessary to withdraw them because of the distressing symptoms of iodism. The patient was quite well for some time before his death from influenzal pneumonia.

On the recognition of the second case as one of oidiomycosis the oral treatment was replaced by the intravenous, and a hypertonic solution of sodium iodid was given. There was an immediate subsidence of the symptoms. At the time of the latest reports the man was perfectly well.

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* Presented before the New York Neurological Society, Dec. 6, 1921.

1. Weed and McKibben: Pressure Changes in Cerebro-Spinal Fluid Following Intravenous Injection of Solutions of Various Concentrations. Experimental Alteration of Brain Bulk, Am. J. Physiol. **48**:512-558, 1919.

2. Farnell, F. J.: Systemic Oidiomycosis with Manifestations in the Central Nervous System, Boston M. & S. J. **176**:771-773, 1917.

The patient in the third case, a girl of 13 years, seen by the examiner last spring, presented cutaneous and bone lesions resembling very closely oidiomycosis, but considered syphilitic by previous practitioners. She had been treated with mercury and iodids by mouth for several months, but made no great progress. Evidently sufficient treatment with iodids had been given to disturb the possibility of obtaining the oidium in pure strain. However, the Wassermann reaction was negative, and the poor response to antisyphilitic treatment led the author to give an intravenous injection of hypertonic sodium iodid. The lesions were healed in forty-eight hours, and the patient remained perfectly well up to date (six months).

Recognizing the value of intravenous hypertonic iodids in the treatment of oidiomycosis, during the past two years at least 400 intravenous injections of hypertonic iodids have been given, and observations have been made on the value of this iodin therapy.

A great deal of attention is being paid to the relation of either organic or inorganic iodin and the thyroid gland. It has been used in vitamin experiments by Emmett, Allen and Sturtevant.[3]

Kendall and Richardson [4] have determined the iodin content in the blood. Osborne [5] has determined the iodin content in the spinal fluid.

In the 1921 volume of "New and Nonofficial Remedies," [6] as accepted by the Council on Pharmacy and Chemistry of the American Medical Association, it is stated in regard to iodin compounds for internal use:

> These are typified by the ordinary iodides. The mechanism of their action is not understood. The most definite results are seen in the rapid absorption of certain inflammatory exudates and especially of the tertiary lesions of syphilis (gummas); lesions of the bone, skin, brain, or other internal organs, disappear under adequate doses of the drug. Likewise in actinomycosis and sporotrichosis the action of iodine as iodide is almost specific. The iodides are not germicidal.
>
> The effect of iodides in arteriosclerosis and aneurysm is probably limited to the absorption of syphilitic deposits in the vessel wall. The iodides do not directly lower blood-pressure. They increase the amount of iodothyrin and may thus have indirect effects on metabolism.
>
> A striking innovation in iodide therapy has been the introduction of compounds of iodine with proteins and fats. The advocates of these organic

3. Emmett, Allen, and Sturtevant: Relation of Vitamines and Iodine to the Size and Development of Tadpoles, J. Biochem. **41:** No. 3, p. 54 (Proceedings).

4. Kendall and Richardson: Estimation of Iodine in the Blood, J. Biochem. **42:**149, 1921.

5. Osborne, Earl D.: Iodin in the Cerebrospinal Fluid with Special Reference to Iodid Therapy, J. A. M. A. **76:**1384-1386 (May 21) 1921.

6. New and Nonofficial Remedies, Chicago, American Medical Association, 1921, p. 150.

combinations assert that they are less irritating to the digestive tract and that they are less inclined to set up the disagreeable symptoms of iodism, for instance, coryza and skin eruptions. Experience confirms in a measure the former claim, but the latter is misleading. Iodism is probably a necessary manifestation of the full physiologic activity of the drug, which is required for the treatment of syphilis and other diseases. If, therefore, a preparation fails to elicit these characteristic symptoms, we conclude that the amount of the drug absorbed is insufficient. Clinical observations show that the organic iodides, in the dosage ordinarily employed, are weaker than the metallic forms.

It is observed that the percentage of iodin is greatest in ammonium iodid, 87.5 per cent.; next in sodium iodid, 84.6 per cent, and next in potassium iodid, 76.4 per cent.

I have used sodium iodid exclusively in my work. Sodium iodid has been used because of the nonactivity of the sodium ion. The preparation is made in from 8 per cent. to 15 per cent (10 per cent. is the usual rate) solutions in distilled water, which is also autoclaved—amount, 100 c.c. This is then boiled and cooled and given by gravity intravenously (made fresh for each treatment). A distinctly hypertonic solution of high concentration is thus produced.

By this method of high concentration and its immediate introduction into the blood stream, in none of the examiner's observations has there been any irritation of the digestive tract. Skin eruptions were observed in one case (no coryza). Coryza (no skin eruptions) was observed in one case.

The patients made such comments as the following: A brassy taste may make itself manifest during the period of injection and from then up to and including three days. This may mean that the iodin is not eliminated completely for about three days. Several patients have referred to the odor of their perspiration which will last for about two days. Within the first twenty-four hours after the introduction of iodin some patients will have a profuse copious stool, dark colored and odorous.

Strange as it may seem, the odor of the perspiration and the copious stool production has occurred only in those cases in my group in which the symptoms were related to alcoholism and in which there were definite indications of cerebral edema.

In patients with syphilis (cerebrospinal) in whom the liver was undoubtedly attacked, the gastro-intestinal complaints and the hypochondriac distress abated soon after treatment. It is a fact that iodin is capable of increasing autolysis, especially in the liver.

Striking results have been observed in those cases in which there is a presumed increase in the bulk of the brain—cerebral thrombosis, cerebral hyperemia, cerebral edema, etc. Weed and McKibben believe that the brain should no longer be considered as incompressible or of

fixed volume, but that it is subject to variation in size. The cranial cavity is fixed in volume and contains a brain, blood and cerebrospinal fluid. Variations in any one may occur, compensation being afforded by an alteration in the volume of one or both of the remaining elements.

Among my patients, one with cerebral hyperemia received relief from headache and ocular symptoms after the introduction of a 10 per cent. solution of iodid. A patient with cerebral edema, cardiovascular, with a convulsion and resultant hemiplegia, received five injections of a 10 per cent. solution at four-day intervals, which was followed by a rapid readjustment of his cerebral disorder.

Patients with cerebrospinal syphilis, of the meningitic and meningovascularis types, appear to respond more rapidly to arsphenamin treatment by receiving occasional injections of 10 per cent. sodium iodid. Iodin being a reducing agent, it probably increases the permeability of the cells of the meninges.

High blood pressure has not been reduced, but a distinct change in the general metabolism of the patients occurs.

COMMENT

1. Do tissue and cell changes occur more rapidly when a highly concentrated solution of sodium iodid is injected directly into the blood stream?

2. Could the same result be obtained by giving a concentrated solution with a bland syrup, by mouth?

3. Does the apparently good result depend on the high concentration or on the intravenous channel of administration?

SUMMARY

Iodin in the form of an iodid can be given in hypertonic form intravenously.

Iodin when injected into the blood stream in hypertonic form has a tendency to reduce the idiosyncrasy toward iodism.

Iodids introduced into the blood stream appear to readjust systemic fungus disturbances (oidiomycosis) very rapidly.

Iodids injected intravenously in concentrated form appear to help materially the action of arsphenamin on the diseased tissues and cells.

Iodids introduced into the blood stream in hypertonic solutions probably have some influence on reducing the edema, hyperemia, etc., of the brain in increased brain bulk disorders.

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SEROLOGIC CHANGES IN NEUROSYPHILITIC PATIENTS DURING A PERIOD OF NONTREATMENT*

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AND

P. H. CARROLL

MENDOTA, WIS.

A selected group of paretic patients was studied during a period of over six months, during which time no specific medication was administered. This to our knowledge has not been done before. The patients selected for this investigation had not received any treatment for several years. All cases were under constant supervision, and careful periodic mental and physical examinations were made. The patients were carefully chosen according to their mental and physical findings and had undoubted cases of paresis.

In paretic patients, laboratory findings frequently change during a period of specific treatment, and such changes are quite generally accepted as a result of the treatment. Our serologic investigation was made by a method which we believe gives constant results. This method shows degress of positive reaction and hence permits a comparison of findings over an extended period.

The laboratory procedures were: Wassermann titration of blood serum by a titration method developed by Dr. W. F. Lorenz, Director of the Wisconsin Psychiatric Institute (personal communication to be reported later); Wassermann titration of cerebrospinal fluid by the same method; a cell count; Lange's colloidal gold test, and the Ross-Jones and Noguchi tests for globulin.

In the Wassermann tests the antihuman system was employed throughout. Fresh pooled guinea-pig serum was used as complement; serum from immunized rabbits was used as amboceptor. Noguchi's acetone-insoluble extract of human heart muscle was used as antigen. The antigen used was neither anticomplementary nor hemolytic in ten times the amount used in the test and the dose employed was 0.1 c.c. of a 10 per cent. dilution which was four times the amount required to give a positive reaction with pooled syphilitic serum.

TECHNIC OF THE WASSERMANN TITRATION ON BLOOD SERUM

Test-tubes are set up in four rows as follows:

Row A: To determine the amount of native complement present in the serum. Measured amounts of diluted unheated serum are employed throughout.

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* From the laboratories of the Wisconsin Psychiatric Institute.

Row B: Serum plus 1 unit complement.

Row C: Antigen plus 1 unit complement.

Row D: Antigen plus 2 units complement.

After withdrawal the blood is allowed to clot at room temperature and then placed in the icebox for twenty-four hours. After centrifugation the serum is prepared in the following dilutions 2.5, 5, 10, 20 and 50 per cent., and 0.1 c.c. of each dilution is used in the test. Table 1 illustrates the method.

TABLE 1.—WASSERMANN TITRATION OF BLOOD SERUM

D	1 c.c. of 2.5% serum Antigen 2 units complement	1 c.c. of 5% serum Antigen 2 units complement	1 c.c. of 10% serum Antigen 2 units complement	1 c.c. of 20% serum Antigen 2 units complement	1 c.c. of 50% serum Antigen 2 units complement
C	1 c.c. of 2.5% serum Antigen 1 unit complement	1 c.c. of 5% serum Antigen 1 unit complement	1 c.c. of 10% serum Antigen 1 unit complement	1 c.c. of 20% serum Antigen 1 unit complement	1 c.c. of 50% serum Antigen 1 unit complement
B	1 c.c. of 2.5% serum 1 unit complement	1 c.c. of 5% serum 1 unit complement	1 c.c. of 10% serum 1 unit complement	1 c.c. of 20% serum 1 unit complement	1 c.c. of 50% serum 1 unit complement
A	1 c.c. of 2.5% serum	1 c.c. of 5% serum	1 c.c. of 10% serum	1 c.c. of 20% serum	1 c.c. of 50% serum

One c.c. of 1 per cent. corpuscle suspension is added to each tube and the series is incubated for one hour at 37.5 C. Amboceptor is then added, and the series is again incubated with frequent shaking of the tubes during the last incubation period.

TECHNIC OF THE WASSERMANN TITRATION IN THE CEREBROSPINAL FLUID

Row A: Control plus 1 unit of complement.

Row B: Antigen with 1 unit of complement added.

Row C: Antigen with 2 units of complement added.

Since cerebrospinal fluid does not contain native complement only three rows of tubes are necessary, one for control and two for antigen. The cerebrospinal fluid is used in the following dilutions: 5, 10, 20 and 50 per cent.; of these 0.1 c.c. is used in each tube. A series containing 0.1, 0.3 and 0.5 c.c. of undiluted fluid is included in the test.

Table 2 illustrates the set up for the titration in the spinal fluid.

One c.c. of 1 per cent. corpuscle suspension is added to each tube, the series incubated for one hour at 37.5 C., and at the end of the first hour's incubation, an amboceptor unit is added, and the series is again incubated for an hour with frequent shaking of tubes.

TECHNIC OF DAILY TITRATIONS FOR COMPLEMENT UNITS

A series of four rows of test-tubes are set up as follows:

Row A contains 1 c.c. of 1 per cent. corpuscles with a dose of amboceptor, and varying amounts of 10 per cent. fresh pooled guinea-pig serum.

Row B: Same as row A with the addition of nonactive negative serum.

TABLE 2.—WASSERMANN TITRATION IN THE CEREBROSPINAL FLUID

	1 C.c. Spinal Fluid in Dilutions of				Undiluted Spinal Fluid		
	5%	10%	20%	50%	0.1 C.c.	0.3 C.c.	0.5 C.c.
C	Antigen 2 units complement	Antigen 2 units complement	Antigen 2 units complement	Antigen 2 units complement	Antigen 2 units complement	Antigen 2 units complement	Antigen 2 units complement
B	Antigen 1 unit complement	Antigen 1 unit complement	Antigen 1 unit complement	Antigen 1 unit complement	Antigen 1 unit complement	Antigen 1 unit complement	Antigen 1 unit complement
A	1 unit complement	1 unit complement	1 unit complement	1 unit complement	1 unit complement	1 unit complement	1 unit complement

TABLE 3.—DAILY TITRATIONS FOR COMPLEMENT UNITS

D	0.1 c.c. antigen 0.04 c.c. serum 1 c.c. 1% corpuscles 1 unit amboceptor 0.05 c.c. 10% complement	0.1 c.c. antigen 0.04 c.c. serum 1 c.c. 1% corpuscles 1 unit amboceptor 0.1 c.c. 10% complement	0.1 c.c. antigen 0.04 c.c. serum 1 c.c. 1% corpuscles 1 unit amboceptor 0.15 c.c. 10% complement	0.1 c c. antigen 0.04 c c. serum 1 c c. 1% corpuscles 1 unit amboceptor 0.2 c.c. 10% complement
C	0.1 c.c. antigen 1 c.c. 1% corpuscles 1 unit amboceptor 0.05 c.c. 10% complement	0.1 c.c. antigen 1 c.c. 1% corpuscles 1 unit amboceptor 0.1 c.c. 10% complement	0.1 c.c. antigen 1 c.c. 1% corpuscles 1 unit amboceptor 0.15 c.c. 10% complement	0.1 c c. antigen 1 c.c. 1% corpuscles 1 unit amboceptor 0.2 c.c. 10% complement
B	0.04 c.c. neg. serum 1 c.c. 1% corpuscles 1 unit amboceptor 0.05 c.c. 10% complement	0.04 c.c. neg. serum 1 c.c. 1% corpuscles 1 unit amboceptor 0.1 c.c. 10% complement	0.04 c.c. neg. serum 1 c.c. 1% corpuscles 1 unit amboceptor 0.15 c.c. 10% complement	0.04 c.c. neg. serum 1 c.c. 1% corpuscles 1 unit amboceptor 0.2 c.c. 10% complement
A	1 c.c. 1% corpuscles 1 unit amboceptor 0.05 c.c. 10% complement	1 c.c. 1% corpuscles 1 unit amboceptor 0.1 c.c. 10% complement	1 c.c. 1% corpuscles 1 unit amboceptor 0.15 c.c. 10% complement	1 c.c. 1% corpuscles 1 unit amboceptor 0.2 c.c. 10% complement

Row C: Same as row A with one antigen unit.

Row D: Same as row A with antigen unit and nonactive negative serum.

Table 3 illustrates the set-up for the daily complement titration.

One c.c. corpuscles 1 per cent. and 1 dose amboceptor is added to each tube.

Two titrations are made. In one the amboceptor is added immediately, in the other titration the amboceptor is added after one hour's incubation—after the addition of amboceptor in both of these titrations

the series is incubated at 37.5 C. for one hour. The tube showing complete hemolysis with the least amount of complement is considered as the unit for that day. The result in the titration made with immediate addition of amboceptor must not vary beyond one tube from the result obtained with the titration in which the amboceptor was added at the end of one hour. Likewise the unit obtained with the hemolytic system must not vary beyond one step from that in which antigen or antigen and negative serum are present. This procedure serves as a careful check on any anticomplementary properties that may develop in a stock antigen.

TABLE 4.—Findings in Wassermann Titration of Blood

	1 C.c. Serum in Dilutions of				
	2.5%	5%	10%	20%	50%
Initial observation:					
D	H	H	H¼	+	+
C	H	H¼	+	+	+
A	+	+	H¼	H½	H¾
After an interval of 1 week:					
D	H	H	H	+	+
C	H	H	+	+	+
A	+	+	+	+	H¼
After an interval of 2 weeks:					
D	H	H	H	+	+
C	H	H	H¾	+	+
A	+	+	H¼	H½	H¾
After an interval of 3 weeks:					
D	H	H	H	+	+
C	H	H	H	+	+
A	+	+	H¼	H¼	H½
After an interval of 4 weeks:					
D	H	H	H¼	+	+
C	H	H	+	+	+
A	+	+	+	+	H¼
After an interval of 5 weeks:					
D	H	H	H	+	+
C	H	H½	H¼	+	+
A	+	+	+	H½	H¾
After an interval of 7 weeks:					
D	H	H	H¾	+	+
C	H	+	+	+	+
A	+	+	+	H¼	H½
After an interval of 9 weeks:					
D	H	H	+	+	+
C	H	H¾	+	+	+
A	+	+	+	H¾	H
After an interval of 13 weeks:					
D	H	H½	H¼	+	+
C	H	+	+	+	+
A	+	+	+	H¼	H½
After an interval of 17 weeks:					
D	H	H	H½	+	+
C	H	H	+	+	+
A	+	+	+	H¼	H½
After an interval of 31 weeks:					
D	H	H	H½	+	+
C	H	H	+	+	+
A	+	+	+	H¼	H½

REPORT OF CASE

History.—A white man, 38 years old, single, a manufacturer of restraints, had a syphilitic infection nine years ago, after which he took mercury pills for two years. The onset was gradual, characterized by delusions of persecution three years prior to admission.

The patient was admitted to the Wisconsin State Hospital at Mendota, Wis., March 11, 1918.

Mental Examination.—On admission the patient was elated, exalted and grandiose. During examination he was very irritable, restless, easily annoyed and showed poverty of ideas and delusions of persecution. Emotional exaltation, marked intellectual deterioration and defective memory were present.

Physical Examination.—The patient was apparently strong and healthy. His thyroid was palpable, his heart and lungs negative. The blood pressure was: systolic 134 and diastolic 78. He weighed about 120 pounds (54 kg.).

TABLE 5.—Findings in Wassermann Titration of Spinal Fluid

	1 C.c. Spinal Fluid in Dilutions of				Undiluted Fluid		
	5%	10%	20%	50%	0.1 c.c.	0.3 c.c.	0.5 c.c.
Initial observation:							
C	H	H	H	+	+	+	+
B	H	H	+	+	+	+	+
After an interval of 2 weeks:							
C	H	H	H	H¼	+	+	+
B	H	H	+	+	+	+	+
After an interval of 5 weeks:							
C	H	H	H	+	+	+	+
B	H	H	H	+	+	+	+
After an interval of 9 weeks:							
C	H	H	H	H½	+	+	+
B	H	H	H	+	+	+	+
After an interval of 13 weeks:							
C	H	H	H	H½	+	+	+
B	H	H	H½	+	+	+	+
After an interval of 17 weeks:							
C	H	H	H	H½	+	+	+
B	H	H	H½	+	+	+	+
After an interval of 30 weeks:							
C	H	H	H	H	+	+	+
B	H	H	H	+	+	+	+

TABLE 6.—Results of Colloidal Gold, Cell Counts and Globulin Tests

	Colloidal Gold Test	Cell Count	Ross-Jones and Noguchi Tests
Initial test	5555555210	35	+
After an interval of 2 weeks	5555555210	35	+
After an interval of 5 weeks	5555554210	33	+
After an interval of 9 weeks	5555432100	21	+
After an interval of 13 weeks	5555542100	30	+
After an interval of 17 weeks	5555553100	28	+
After an interval of 30 weeks	5555554100	21	+

Neurologic Examination.—The patient had no anosmia. His pupils were irregular, miotic and equal; they reacted to accommodation and sluggishly to light. Marked tremor of the facial muscles, tongue and hands was present. The tendon reflexes were hyperactive. The superficial reflexes were normal. Pathologic toe findings were present on both sides; there were no definite sensory changes. A slight Romberg sign was present. The gait was good, speech slurring and slow. Writing was tremulous; there were no omissions.

Laboratory Findings on Admission.—These were: Blood Wassermann test ++++, cerebrospinal fluid test ++++; colloidal gold test 5555541000; Ross-Jones test, positive; Noguchi test, positive; 29 cells.

Serologic Findings.—Wassermann titration of the blood was made.

In recording the findings over a period of observation only the results of column A (titration for native complement), column C (titration with one unit of complement) and column D (with two units of complement), will be included, it being understood that column B (hemolytic system control) will show complete hemolysis in all tubes. The results are recorded by the following scheme:

H indicates complete hemolysis.
H ¾ indicates hemolysis of approximately ¾ of the corpuscles
H ½ indicates hemolysis of approximately ½ of the corpuscles.
H ¼ indicates hemolysis of approximately ¼ of the corpuscles.
+ indicates no hemolysis (complete inhibition).

Color comparison was used as the guide in determining the grades of partial hemolysis.

Wassermann titration of spinal fluid was also made. Row B shows the result with one unit of complement and row C with two units. The control row is omitted in Tables 5 and 6.

Nine cases in all were similarly studied and eight of the nine gave findings which were uniformly positive in both the blood and cerebrospinal fluid.

One case (No. 109) was diagnosed as paresis in 1914. The patient received antisyphilitic treatment during 1914 and 1915, and the condition might be regarded as clinically arrested. The serologic examinations were negative in this case throughout the period of observation.

CONCLUSIONS

The laboratory findings in a series of nine cases of late neurosyphilis remained practically unchanged during a seven month period of nontreatment.

EPIDEMIC ENCEPHALITIS WITH MYELITIS*

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The spinal cord has been little implicated in epidemic encephalitis and I know of no case except the one I now report in which intense inflammation was found as low as the lumbar region. The case reported in this paper is interesting in its symptoms suggesting brain tumor; in its pathology presenting a myelo-encephalitis; and in the resemblance of its lesions to those of multiple sclerosis, affording support for the theory of the inflammatory origin of the latter disease.

Nuclear stains in this case yielded different findings from those of multiple sclerosis in the intense perivascular infiltration and the presence of many fatty granular or gitter cells in the tissues, and yet it seems reasonable to suppose that had death been delayed a year or more the structure of these sclerotic areas would have resembled that of the areas in multiple sclerosis. The Weigert hematoxylin stain gives findings closely resembling the sclerotic patches of multiple sclerosis, as shown by the illustrations. If we accept the view that fatty granular cells are derived from glia cells and cells in the walls of the blood vessels, as Schröder [1] maintains, we may expect these cells to assume again more the appearance of glia cells in lesions of long duration.

The spinal cord has been found to be implicated in epidemic encephalitis by Harbitz and by Winkelman and Weisenburg.[2] In the case reported by the latter the lesions in the cord were not intense, they were similar in character to those in the brain, and the spinal cord below the middle or lower thoracic region was entirely normal. There were no sclerotic patches.

REPORT OF CASE

History.—I. R. B., a girl, aged 15 years, was admitted to the University Hospital, April 20, 1921. About Feb. 1, 1921, the father noticed that she walked with her head farther forward than usual. She vomited a few times about the first of March and complained of dizziness and headache. The vomiting usually occurred a short time after eating. She complained of the dizziness and headache all the time she was standing. She began to stagger a little in walking, early in March, and appeared as though she might be intoxicated. Later

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* Read before the Philadelphia Neurological Society, Feb. 24, 1922.

1. Schröder: Berl. klin. Wchnschr. **54**:714, 809, 1917.

2. Winkelman, N. W., and Weisenburg, T. H.: A Case of Myoclonic Lethargic Encephalitis with Unusual Pathologic Manifestations, Phila. Neurol. Soc. Trans., Arch. Neurol. & Psychiat. **5**:224 (Feb.) 1921.

in March it became necessary for her to hold on to objects in walking. Her father noticed that she was becoming dull during the latter part of February, 1921. She did not notice things as she had formerly, and she did not play. She did not seem to understand immediately when spoken to. She did her work as usual, but when she had nothing to do she sat listless, and this languor persisted.

She was taken to St. Luke's Hospital, Bethlehem, and it is not surprising that a diagnosis of cerebellar tumor was made. She began to have a little difficulty in talking in April; she knew what she wanted to say but had difficulty in forming the words. This difficulty rapidly increased. She began to complain of numbness of the right upper and lower limbs early in April, also of numbness of the tongue and some difficulty in swallowing. Her father thought her whole right side had become weak, but this paralysis is uncertain. She had had no convulsions. She still had slight headache when she came to the

Fig. 1.—Weigert medullary sheath stain of a section of the pons. Sclerotic patches are sharply defined on one side of the raphé, while on the other side the degeneration is more diffuse.

University Hospital. Constipation had become marked. She had no dyspnea or palpitation. She had difficulty in passing the urine, and at times had to be catheterized. Menstruation was regular up to the time she came to the hospital. A severe attack of scarlet fever had left her deaf in the left ear.

Examination.—When she entered the University Hospital she was lethargic; she answered when spoken to but became tired after answering a few questions, and did not notice what was going on about her. Her articulation was poor. Respiration was normal. The tongue was protruded poorly and deviated to the left. There was little movement of the eyes laterally, but some move-

ment upward with nystagmus, and better downward movement. There probably was bilateral ptosis.

Biceps tendon reflexes were not obtained and the triceps tendon reflexes were slight. The left patellar reflex was moderately exaggerated, and the right was somewhat diminished. Both Achilles reflexes were exaggerated, and bilateral ankle clonus was obtained. In the Babinski reflex there was upward movement of the toes on each side.

The handgrasp was weak on each side. The patient was unable to straighten her fingers, and they were held slightly flexed.

Dr. B. F. Baer found the optic disks sharply marked and the eye-grounds negative.

Notes obtained from Dr. Francis J. Dever, Bethlehem, stated that she had been admitted to St. Luke's Hospital in April, 1921, with nausea, vomiting and headache. The blood Wassermann reaction was negative. An examination of

Fig. 2.—Section through the medulla oblongata.

the spinal fluid revealed 3 cells per cubic millimeter. The Wassermann test of the fluid was negative, and Fehling's solution was reduced. The right internal rectus muscle was paralyzed and the external rectus was weak. Similar muscle changes were obtained in the left eye. The only fundus change was slight pallor of the temporal side of the disks.

Dr. A. B. Hamilton had found partial ptosis of each upper lid, slight nystagmus on looking to the left, marked nystagmus on looking to the right, and marked nystagmus on looking upward. The left ear was completely dead. Biceps and triceps reflexes were slightly exaggerated, and marked dysmetria was present on each side in the finger to nose test. Diadokokinesia was normal. The upper and lower limbs were not believed to be paralyzed. The gait was of the cerebellar type, and Romberg's sign was marked. Tactile and pain sensations were normal.

On April 24 her mentality was too feeble to permit careful examination, but it was possible to determine that the left lower facial nerve supply was completely paralyzed, and that the upper supply was weak, though she could close the left eyelids. She understood a simple command, such as to show the teeth, and performed this movement several times. She gradually became worse and died May 5, 1921.

The important symptoms in this case were: headache, vertigo, vomiting, mental impairment and lethargy, numbness and possibly weakness of the right limbs, nystagmus, dysmetria, incoordination of gait and station, exaggeration of the tendon reflexes of the lower limbs, the Babinski sign, paralysis of the left seventh nerve and ocular palsies.

A diagnosis of cerebellar tumor was not unreasonable, and certain symptoms suggested multiple sclerosis; but the case appeared to me to be one of epidemic encephalitis, and microscopic study of the tissues proved that this diagnosis was correct.

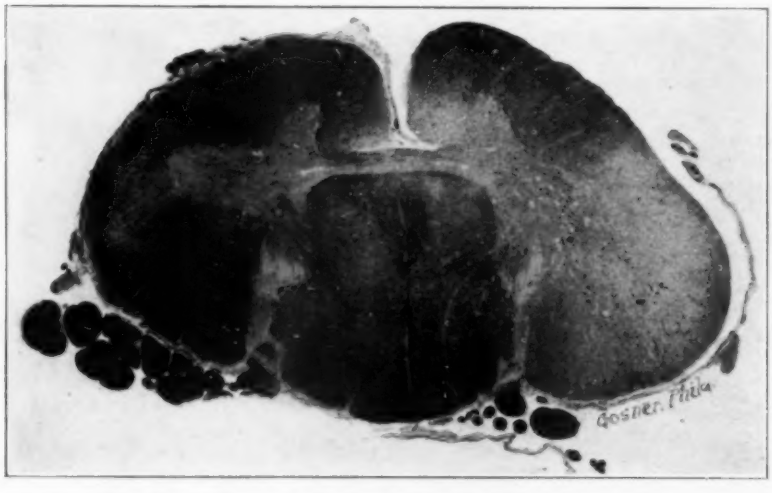

Fig. 3.—Section about the first thoracic segment.

Microscopic Examination.—Sections from the frontal and occipital lobes and the paracentral lobule showed nothing abnormal. The inflammation was of moderate intensity in the basal ganglions, but it was intense in the cerebral peduncles. Here some sclerotic patches were found by the Weigert hematoxylin stain, but they were not so numerous as in the pons and medulla oblongata. The cerebral peduncles were the highest level at which these patches were seen. The characteristic lesions of epidemic encephalitis were pronounced in the cerebral peduncles—the perivascular infiltration of lymphocytes and plasma cells and the diffusion of these cells throughout the tissues, especially in the gray matter below the aqueduct of Sylvius. Occasionally an area was found containing many spaces from which the nerve fibers had disappeared, resembling the areas so common in severe anemia of the spinal cord; in some of these the cellular infiltration was only of moderate intensity.

The pons showed numerous sclerotic patches by the Weigert hematoxylin stain (Fig. 1), resembling closely those of multiple sclerosis as presented by this stain. I have sections from cases of multiple sclerosis stained by this method which would be hard to distinguish from these of epidemic encephalitis. These areas were in places sharply defined like those considered so characteristic of multiple sclerosis. I have found nothing like them in any other case of epidemic encephalitis except a single area in the cerebral peduncles in one case, nor have I seen such areas described in the literature. With thionin or a nuclear stain like Delafield's hematoxylin they showed intense cellular infiltration, consisting of lymphocytes, plasma cells, glia cells and large globular cells with a small nucleus (fatty granular cells or gitter cells), differing from the areas of acute or subacute multiple sclerosis but resembling them. The characteristic lesions of epidemic encephalitis were intense in the pons and medulla oblongata. I have not seen more intense perivascular cellular infiltration in any case of this disease. The medulla oblongata was affected as severely and in the same way as the pons (Fig. 2). The cerebellum showed nothing striking.

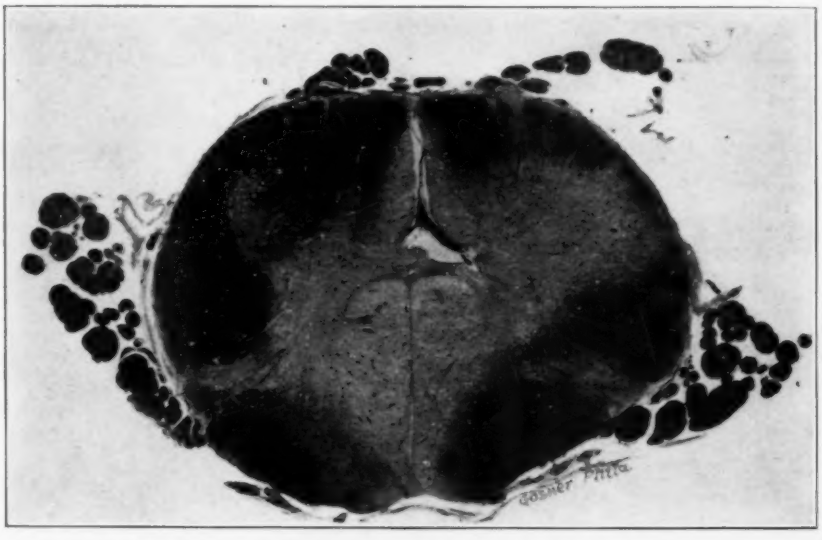

Fig. 4.—Section from the lumbar region showing sharply defined sclerotic areas.

The lower cervical and upper thoracic region (Fig. 3) presented marked sclerotic patches, and they consisted largely of proliferated neuroglia. There was moderate cellular infiltration in these areas but not so intense as in the lumbar region. The degeneration was not confined to any system of fibers, as would be the case in secondary degeneration.

The midthoracic region appeared to be normal, except that in the lateral columns there were one or two areas of very loose neuroglia tissue. This was interesting in view of the great implication of the cervical and lumbar regions, but axis cylinders escaped degeneration, at least partly. It is possible that the spinal inflammation was not of sufficient duration for the development of secondary degeneration.

The midlumbar region (Fig. 4) showed a condition similar to that of the cervical region, but here there was a pronounced cellular infiltration of the gray matter of much the same intensity as in the pons and medulla oblongata, remarkable in its occurrence so low in the cord. I have not seen so grave an implication of the lumbar region in epidemic encephalitis.

ACTINOMYCOSIS OF THE CENTRAL NERVOUS SYSTEM *

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Actinomycosis, while a relatively uncommon disease, has been recognized for years. As early as 1841, it was known to affect man. Some years later Ponfick [1] reported the first two cases of actinomycosis of the brain.

The frequency with which the central nervous system is involved in actinomycosis is difficult to determine. Osler,[2] Lewandowsky,[3] Oppenheim [4] and others report isolated cases and call attention to its rarity. Ruhräh,[5] in 1899, collected sixty-five cases of actinomycosis, nine with involvement of the nervous system. Von Baracz,[6] in 1903, reported sixty cases, in no instance showing involvement of the central nervous system. Cranwell,[7] in an exhaustive investigation in South America, fails to mention cerebrospinal invasion.

The impression gained would be that the central nervous system is markedly free from actinomycotic involvement. However, in a careful review of the literature forty-eight cases of actinomycosis with involvement of the central nervous system have been collected. Sagredo,[8] in 1919, collected twenty cases; they are here included.

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* Presented before the Neurological Surgical Society, Mayo Clinic, Rochester, Minn., Nov. 5, 1921.

1. Ponfick, E.: Die Actinomykose des Menschen, eine neue Infectionskrankheit, Berlin, Hirschwald, 1882, 138 pp.

2. Osler, W.: The Principles and Practice of Medicine, New York, Appleton, 1917, 1225 pp.

3. Lewandowsky, M.: Handbuch der Neurologie, Berlin, J. Springer, 1910, **3**: Pt. 2, p. 216.

4. Oppenheim, H.: Lehrbuch der Nervenkrankheiten für Aerzte und Studierende, Berlin, S. Karger **2**: 1158, 1913.

5. Ruhräh, J.: Actinomycosis in Man, with Special Reference to the Cases Which Have Been Observed in America, Ann. Surg. **30**:417-451; 604-631, 721-746, 1899.

6. Von Baracz, R.: Report of Sixty Cases of Actinomycosis, Ann. Surg. **37**:336-340, 1903.

7. Cranwell, D. J.: Contribucion al estudio de la actinomicosis humana; su frecuencia en la Republica Argentina, Buenos Aires: Hermanos, 1904, 193 pp.

8. Sagredo, N.: Actinomycose du cerveau, Rev. méd. de la Suisse Rom. **39**:478-492, 505-516, 1919.

In this group of forty-eight cases, males were more frequently affected than females. The average age of the patients was about 30 years. The duration of the disease ranged from six months to three years; it rarely lasted more than one year. The majority of cases followed actinomycosis of the jaw, the central nervous system being involved by contiguity. The disease may occur primarily in the brain, though this is rare. Meningeal involvement is the usual form, abscess formation is fairly common, while tumor formation is unusual. Involvement by way of the lymphatics has never been noted.

Musser,[9] Sagredo and Corinth,[10] Howard[11] and others have reported on the spinal fluid. Sagredo obtained 70 per cent. of polymorphonuclears and 30 per cent of lymphocytes, while Corinth reports purulent fluid containing streptococci. No mention is made of the early findings nor of when the purulent fluid developed.

Headache played only a slight part in the early symptomatology of the disease, although in several cases marked neuralgic pains over the swelling, especially when it was around the face, were noted. Convulsive attacks and palsies were not uncommon but presented nothing of diagnostic value. As a rule, the disease had developed to a marked degree before the diagnosis was made. In several cases the diagnosis was made only after finding sulphur granules postmortem. In spite of the severe infection, the temperature was usually low. This afebrile course of the disease was noted as early as 1887 by Bollinger.[12]

SEVENTY-FOUR CASES OF ACTINOMYCOSIS IN THE MAYO CLINIC

The cases reviewed for this study were observed in the Mayo Clinic. In all a positive diagnosis was made microscopically by finding the typical sulphur granules in the pus (Fig. 1). Eighty per cent. of the patients were males, the greater number young adults. The youngest patient was 15, the oldest 71. A large percentage were farmers or were connected with rural life. A history of direct exposure to diseased cattle was rarely obtained. The primary location, so far as could

9. Musser, J. H.; Pearce, R. M., and Gwyn, N.: Two Cases of Streptothrical Infection: Demonstration of a Streptothrix, or Branched Tubercle Bacillus in the Sputum, Trans. Assn. Am. Phys. **16**:208-216, 1901.

10. Corinth, Margarete: Ueber einen Fall von Aktinomykose des Ganglion Gasseri, Deutsch. Ztschr. f. Nervenh. **65**:155-168, 1920.

11. Howard, W. T., Jr.: Actinomycosis of the Central Nervous System, with the Report of a Case Due to an Unidentified Member of the Actinomyces Group, J. Med. Res. **9**:301-323, 1903.

12. Bollinger, O.: Ueber eine neue Pilzkrankheit beim Rinde, Centralbl. f. d. med. Wchnschr. **34**:789-792, 1887.

be determined, was faciocervical in fifty-four patients, abdominal in sixteen, pulmonary in three and cutaneous in one.

Twenty-six patients are known to be living, thirty cannot be traced, and eighteen are dead. Of the twenty-six living, the majority have fully recovered and have been well for from five months to three years. The location of the actinomycosis in the eighteen patients who died with the disease was abdominal in eight, faciocervical in five, pulmonary in three and abdominopulmonary in two.

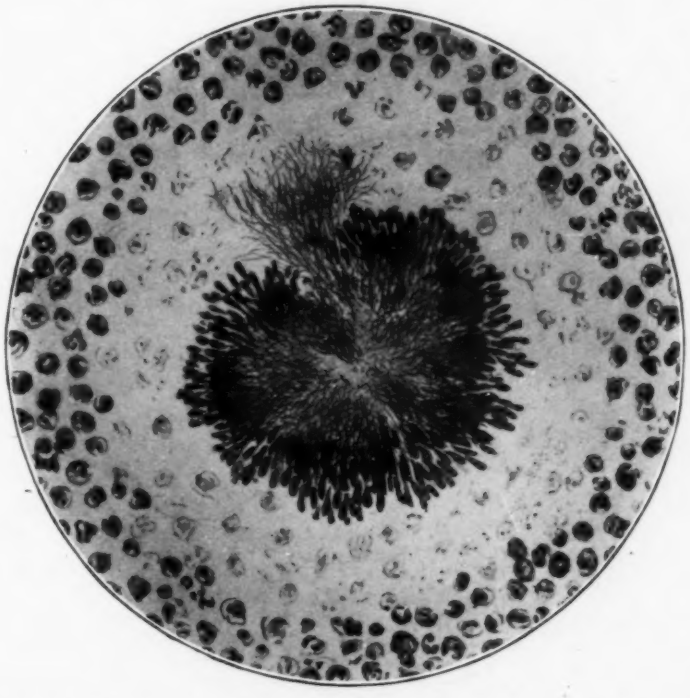

Fig. 1.—Crushed sulphur granule showing ray-fungus, cortical and medullary zones. The cortical zone is made up of club-shaped hyphae; the medullary zone, of filamentous fibers.

There was positive evidence of the involvement of the central nervous system in five of the eighteen patients who died of actinomycosis; the evidence was confirmed by necropsy in two. Two patients with signs of involvement of the central nervous system are alive (Cases 4 and 6 in the table). Two other patients had signs of involvement of the central nervous system, but the subsequent course is not known. These two patients are not included in the series. Undoubtedly other patients in the series of seventy-four had involvement of the central nervous system, but unfortunately the cause of death could not be

determined. The two patients who came to necropsy presented fairly typical pictures. The histories are reported herewith.

REPORT OF CASES

CASE 1 (A 254829).—A man, aged 41 years, was examined Jan. 3, 1919. His personal history was negative, except for tonsillitis in August, 1918, when he had first noticed increasing difficulty in opening his mouth. He complained of pain in the right temporal region, most severe at night.

Examination revealed ankylosis of the jaw, deafness of the right ear, which came on with the tonsillitis, and an unerupted upper right third molar. The results of the neurologic examination were negative. An indeterminate diagnosis was made, and Jan. 8, 1919, roentgen-ray treatments not having caused any apparent change, the jaw was stretched and the upper third molar extracted.

Fig. 2 (Case A 326059).—Section of the spinal cord showing marked meningitis with ependymitis.

The swelling in the region of the right parotid gradually increased, and February 24 a phlegmon over the angle of the jaw was drained. The condition spread and not until one month later, when a phlegmon in the right cervical region was drained and sulphur granules discovered in the pus, was the diagnosis of actinomycosis made.

The patient gradually became weaker, no benefit having been experienced from radium treatment. A second neurologic examination, April 8, revealed paralysis of the right external rectus, nystagmus to the right, hypesthesia and hypalgesia over the right fifth nerve area with weakness of the masseter, temporal and pterygoid muscles. All deep reflexes were exaggerated, the Kernig sign positive and the plantar reflexes flexor. The neck was rigid. The mental state was unimpaired. A diagnosis of low grade meningitis was made. Spinal puncture disclosed a cloudy, thick, purulent fluid; the cells were not counted, but pus cells predominated. No organisms were found and no actinomycetes or sulphur granules. The neck gradually became stiffer; the knee reflexes were difficult to obtain. The right seventh nerve also showed considerable weakness. The patient died April 22, 1919, after being semiconscious for two days.

Necropsy revealed an extensive actinomycetic granuloma of the right parotid region with erosion of the petrous portion of the right temporal bone and basilar and spinal leptomeningitis. The exudate was yellow. A sinus could be traced from the parotid region through the thick portion of the temporal bone into the middle cerebral fossa. Cultures from the spinal fluid gave short chain streptococci but no ray fungi.

Sections were made from various areas of the brain and cervical cord and stained with hemotoxylin-eosin, thionin, Cajal's and Gram's stains. Sections from the vertex showed moderate lymphocytic infiltration of the pia. The cortex showed no marked change; there was no blood vessel infiltration. Gram's stain was negative for this region. The basal portions showed extensive meningeal infiltration, chiefly mononuclear. The process was confined almost entirely to the membranes, the cortical substance remaining relatively free. In the membranes of the right pons a typical ray fungus with marked polymorphonuclear infiltration was found. Gram's stain in every case appeared to be elective, and the growth might have been overlooked if only the usual stains had been employed. Sections of the cord showed similar infiltration of the membranes, predominantly lymphocytic, but no ray fungus.

CASE 2 (A 326059).—A man, aged 25 years, a farmer, was examined Sept. 11, 1920. In February, 1920, he had noticed slight drooping of the right eyelid, with ringing and deafness in the right ear. Some time prior to this he had sustained a small cut over the right eye by a whip. Two weeks later burning pain and numbness in the right leg made walking difficult. About June 1, 1920, he began to experience pain in the right frontal and temporal regions, with swelling in the temporal area.

Examination, July 28, revealed swelling of the right temporal region with exophthalmos of the right eye. The patient was somewhat emaciated. There were blurring of both fundi, deafness in the right ear, diminution of sensation over the right supra-orbital region, and slight weakness of the right leg. July 31, pus containing actinomycetes was drained from a small abscess over the right upper lid. The spinal fluid report was: positive Nonne reaction; 1,889 polymorphonuclear leukocytes, 587 small lymphocytes, and Lange's test 0111221000. Because of the headache and blurring disks a decompression operation was performed August 10. The findings were negative.

The patient was dismissed from observation and returned September, 1920, when the spinal fluid showed 841 polymorphonuclears, 128 lymphocytes and no granules; his temperature was 100 F.; he had no clinical signs of meningitis. He gradually became worse; emaciation was marked; he passed into a mild state of confusion with muttering delirium.

September 26, the patellar and Achilles reflexes could not be obtained; there was no Babinski sign and only moderate rigidity of the neck. Until the day of death no paralysis occurred, although the right leg was weak. At times headache was very severe. The patient died Sept. 28, 1920.

Necropsy revealed the scar of operation, but no infection or growth. The membranes were adherent at the right orbital ridge, and a thick layer of gelatinous, greenish-yellow pus surrounded the area and covered the entire basal surface. Throughout the pus numerous pin-point granules could be seen. The point of infection was apparently at the orbital ridge, where a spot the size of a pencil head could be distinguished and from which a sinus led directly into the orbital space where the pus was very thick and tenacious. The entire cord was bathed in thick, greenish yellow pus, most marked in the caudal region.

ACTINOMYCOSIS OF THE CENTRAL NERVOUS SYSTEM—SEVEN CASES

Case	Sex Age	Occupation	Primary Focus	Duration of Symptoms, Months	Neurologic Signs of Central Nervous System Involvement	Headache	Temperature	Mental Symptoms	Lymphocytes	Spinal Fluid	Diagnosis	Remarks
130341	M 34	Farmer	Chest	18	General weakness, tremor and increased reflexes; repeated convulsive attacks	Moderate	99?	Terminal mental apathy	11,000 16,000	Negative	Actinomycosis, probably abscess	Patient died at home; no necropsy
254829	M 41	Farmer	Jaw	9	Cranial nerve palsy, choked disks, increased deep reflexes, rigid neck and positive Kernig's sign	Moderate	99 100	Terminal unconsciousness	6,800 15,000	Purulent	Actinomycosis meningitis	Necropsy showed meningitis of both brain and cord
320695	M 38	Farmer	Jaw	8	Cranial nerve palsy, rigid neck and paresthesias	Severe, localized in left frontal to parietal area	Moderate	Mild delirium	9,000 11,800	Nonne +; polymorphonuclears 1,735, lymphocytes 170	Actinomycosis meningitis	Patient died at home; no necropsy
325362	M 20	Farmer	Jaw	?	Proptosis, cranial nerve palsy, rigid, neck, no clinical signs of meningitis	Moderate	.....	.....	6,800	Negative	Probably actinomycosis meningitis	Patient alive
326059	M 25	Farmer	Jaw	8	Cranial nerve palsy, headache, rigid neck, delirium, and numbness in limbs	Severe, localized in temporal area	98 100	Mild delirium	6,900	Nonne +, Lange 0111221000, polymorphonuclears 1,889, lymphocytes 587	Actinomycosis meningitis	Necropsy showed meningitis of both brain and cord
347777	M 24	Farmer	Jaw	7	Cranial nerve palsy, rigid neck, mental torpor	Moderate	99 100	Mild grade delirium	12,000	Negative	Actinomycosis meningitis	Patient died, no necropsy
371666	F 27	Housewife	Jaw	6	Motor and sensory involvement of right fifth nerve and headache; no clinical signs of meningitis	Moderate	99 101	.....	11,000	Wassermann +, Nonne +, polymorphonuclears 92, lymphocytes 400	Actinomycosis meningitis	Patient alive

Sections were taken from the brain and cord and stained with hematoxylin-eosin, thionin, Cajal's, Weigert's, and Gram's stains. As in Case 1, the convolutions showed only slight involvement. Sections from the base and especially those taken at the cerebellopontile angle showed severe meningeal infiltration and some polymorphonuclear cells. The reaction was confined chiefly to the membranes in which the fungus was present, although the brain substance showed some cell degeneration and slight blood vessel infiltration.

Sections from the cord showed severe meningitis, with many polymorphonuclear cells. The central canal throughout was filled with polymorphonuclear cells; the exact cause of their occurrence remains uncertain (Fig. 2). The cord substance showed some swelling of the nerve cells with displaced nuclei and moderate blood vessel infiltration, but no abscesses or foci were discovered and no fungi were found in the membranes of the cord.

What has been said of actinomycosis apparently holds true for many other mycotic affections. Ophüls,[13] in 1905, reported thirteen cases of coccidioidal granuloma, three of which showed central nervous system involvement. Rusk and Farnell[14] reported several cases of oidiomycosis with meningitis. Since the work of Gilchrist,[15] over twenty cases of blastomycosis with involvement of the nervous system have been reported. Bassoe,[16] in 1906, first reported a case of blastomycosis with cerebral involvement. Montgomery,[17] in 1917, in a review of blastomycotic infections reported eight cases with brain abscess and several others with involvement of the cord and meninges.

CONCLUSIONS

Actinomycotic involvement of the central nervous system is not rare. The disease process is usually secondary to some other focus in the body, and apparently the meningeal type, in which the infection takes place by contiguity, is most frequent. The course of the disease is chronic, with markedly insidious onset, meningeal involvement occurring without any clinical signs or symptoms. There is some evidence that involvement of the meninges occurs long before the spinal fluid shows evidence of trouble, but the serologic examination usually shows

13. Ophüls, W.: Coccidioidal Granuloma, J. A. M. A. **45**:1291-1296 (Oct. 28) 1905.

14. Rusk, G. Y., and Farnell, F. J.: Systemic Oidiomycosis; a Study of Two Cases Developing Terminal Oidiomycetic Meningitis, with Clinical Notes, Univ. Calif. Pub. Path. **2**:47-58, 1912.

15. Gilchrist, T. C.: A Case of Blastomycetic Dermatitis in Man, Johns Hopkins Hosp. Rep. **1**:269-289, 1896.

16. Bassoe, P.: Disseminated Blastomycosis. Report of a Case Involving the Lungs, Lumbar Vertebrae and Subcutaneous Tissues, with Multiple Abscesses and Fistulae, and Extensive Amyloid Degeneration, J. Infect. Dis. **3**:91-101, 1906.

17. Montgomery, A. H.: Blastomycosis and Sporotrichosis, Surg. Clin. of Chicago **1**:1125-1138, 1917.

a pleocytosis long before there is any clinical evidence of meningitis. It has been suggested that clinical signs of meningitis develop only after secondary infection has occurred. The positive spinal fluid with only slight signs of meningitis is almost pathognomonic. In no other form of severe meningitis do we see such a lack of clinical findings. The ability of these patients to be up and about, presenting few complaints, such as moderate headache, slight rigidity of the neck, and only slight temperature, is practically unknown among other forms of meningitis. During the terminal stage of actinomycosis, the diagnosis is certain, but in the early stages a positive diagnosis on clinical findings alone is practically impossible.

The presence of a chronic phlegmon of the head or neck should indicate the possibility of actinomycosis, and an examination of the spinal fluid and a search for sulphur granules should be made.

The course of the disease is characterized by low grade fever, rarely more than 101 F. and not of the usual septic type, headaches, common and chronic but not unusually severe, terminal delirium, and profound emaciation.

The importance of an early diagnosis in actinomycosis is paramount because of the prognosis. Involvement of the nervous system makes prognosis hopeless. Potassium iodid, arsphenamin and other remedies have been tried, but with uniform failure.

Abstracts from Current Literature

CONTRIBUTION TO THE DOCTRINE OF SENSIBILITY. H. Doebeli, Schweiz. Arch. f. Neurol. u. Psychiat. **9**:75-90, 1921.

Doebeli believes that there is still a large field for the study of sensibility, particularly as it applies to the investigation of disturbances of sensibility that do not reach the field of consciousness. Certainly the largest and physiologically the most interesting group of centripetal impulses do not reach consciousness or are interpreted in a greatly altered manner. A classification of the various known types of sensibility is given, after which Doebeli proceeds at once to his problem: Are there other than the already known forms of irritation capable of exciting the sensory functions of the human organism, such as the ultraviolet rays, the roentgen rays, etc.? He asks the following questions:

1. Is there cutaneous sensibility for moisture? There is no question that rheumatic persons can often foretell with marked precision some change in the weather. When we consider how often moisture is used as a form of therapy, the practical side of this question becomes apparent. Tambroni is the only writer who postulates a so-called "sensibilita igrica," which he went so far as to localize in the brain. His entire doctrine was based on a single case of dementia paralytica. This patient asserted that he could tell when there was moisture in the bed regardless of whether this was obvious or not. On examination of the brain some changes were found in the gyrus hippocampus, from which he assumed that the center of this form of sensibility must be located in this region. He was, as a matter of fact, dealing with illusions and delusions. The marked sensitiveness of the mucous membranes of the mouth, nose and throat to moisture is well known. We are confronted with the question as to whether there is such a form of sensibility present over the cutaneous surface of the body. If so, can any conclusions be drawn relative to the location of these organs of special sensibility?

2. As regards the sensation of itching there is a considerable difference of opinion. For example, Winkler states that itching results from an irritation of Merkel's tactile corpuscles; Thoele, that it is due to a slight irritation of pain fibers; Samberger, that it is the function of specialized organs located among the epithelial cells, particularly in the sheath lying about hair roots. If it is a partial expression of irritation of the organs carrying the sensibility of pain, it should not be present in the analgesic area of certain lesions of the nervous system, such as the lesions of syringomyelia.

3. Is pain elicited on deep pressure over an area of cutaneous analgesia in a case of syringomyelia? In other words, do the tracts carrying cutaneous sensibility and pressure pain sensibility occupy the same position in the cord?

4. One of the forms of deep sensibility is that of joint tenderness. Exactly where arthritic pains arise has not been studied so far as the writer is aware. Does this pain come from the capsule of the joint, from the ligaments, the serous surface, the cartilage, the periosteum, the tendon attachments, or the neighboring muscles; does it arise through pressure, traction or chemical irritation? This type of pain is quite characteristic and can be produced by sub-

luxation of the joint. To what extent is this form of sensibility disturbed in a case of syringomyelia in which cutaneous and deep sensibilities are otherwise interfered with?

5. Where do the receptors lie that subserve the function of so-called position of joint sensibility? Is it a part of deep sensibility as has heretofore been generally accepted, or is it related to cutaneous sensibility, as von Frey and his followers believe?

6. Another form of sensibility which has not been fully investigated is that accompanying local anemia of the tissues, such as can be produced by an Esmarch bandage. This might be called the sensation of blood hunger. Where are the tracts that carry this pain toward the central nervous system? Do they pass through the spinal ganglions and the commissura anterior, similar to impulses of pain, or do they belong to the afferent sympathetic system? This question also applies to the sensation experienced when the blood returns to such an extremity.

7. A contribution of H. Brunschweiler for the first time emphasizes the so-called directional sensibility of the skin and its disturbance in lesions of the parietal region. This directional sensibility can be tested through a light stroking of the fingers over the skin. The question is one of the associations-valence of cutaneous sensibility with the conception of space. In other words, is directional sensibility disturbed over the area of impaired cutaneous sensibility commonly observed in syringomyelia?

8. It has been definitely established that the psychogalvanic phenomenon objectively differentiates hysterical from organic causes of an area of anesthesia. Which afferent tracts produce changes in the skin in the neighborhood of the electrodes in these cases is not known. This subject can be studied to some degree in syringomyelia.

The foregoing problems have been answered through a study of three cases of syringomyelobulbelia.

Case 1.—The patient was one of twins. While going to school the hands were often cold and blue, and the patient had burned himself many times. At about 32 years of age a phlegmon appeared on the right hand, which was followed by progressive weakness in the arms, stiffness of the legs, kyphoscoliosis, atrophy of the shoulder girdle muscles, nystagmus, hemiatrophy of the tongue, syringomyelic paws, and disturbances of touch, pain and temperature sensibility. With the exception of the area supplied by the fifth nerve, pain and temperature were lost to the level of the iliac crests. Tactile and localization sensibility were preserved everywhere with the exception of a small area near the scapula.

Case 2.—The patient was a woman, 39 years of age. At the age of 28, she had painless burns about the right shoulder. At 34 years of age, she had cramps in the right arm and leg and weakness of both arms, nystagmus, atrophy of the shoulder-girdle muscles, fibrillary tremors of the thenar eminence, right pes equinus, increased tendon reflexes, a positive Romberg sign, and the sweat glands ceased functioning. Pain and temperature sense were lost symmetrically below both areas supplied by the fifth to the level of the breasts and the seventh dorsal spine. Tactile and localization sensibilities were normal throughout.

Case 3.—A young woman, aged 25, had scarlet fever with rigidity of the neck at 12 years of age. At 17 years of age, there was a sudden onset of painful cramps; since that time there had been deafness of the right ear,

diplopia and limping. At 22 years of age, she had dyspnea, hoarseness, dysphasia, emaciation, nystagmus, reduced sensibility of the tympanic membrane, paralysis of the soft palate, fibrillary tremors of the pharynx, posticus paralysis, some atrophy of the shoulder girdle muscles, Morvan's syndrome of the right hand and increased tendon reflexes on the left. There was loss of pain and temperature sensibilities below the fifth, with the exception of the left leg and the neighboring saddle area. Tactile sensibility everywhere was normal with the exception of an irregular area over the right shoulder. Pain and temperature sensibility over the right leg was better toward the periphery, but not entirely normal.

Moisture sensibility was investigated in the following manner: Moist or dry air was conducted through a piece of tubing into one end of a box, about the size and shape of the ordinary cigar box. Across the middle of the box was a perforated partition, the purpose of which was to distribute the introduced air more equally. The end of the box opposite the tube was replaced by a batton padding, which permitted close application to the body surface. Near the padding was a hole for the introduction of a thermometer. A hygrometer was suspended in the box near the same end. In carrying out the test, the padded end was placed firmly against the area of skin to be investigated. Dry or moist air was introduced as desired, and the temperature, moisture content and duration of application recorded. The experiment was carefully checked so that all associated factors, such as noise from the source of air supply, could be eliminated. The moisture content in these experiments ranged from 34 to 100 per cent., the temperature from 35 to 54 C., and the duration from ten seconds to seventeen minutes. The subjects were five normal persons, aged 22 to 35 years, one of them an experienced hydrotherapist, a patient with sciatica, 44 years of age, one with muscular rheumatism, 30 years of age, a patient with arthritis, 62 years of age, another with arthritis deformans, 40 years of age, one with rheumatic lumbago, 38 years of age, and one with syringomyelia, 30 years of age.

The result of these experiments was the observation that different degrees of moisture cannot be differentiated when applied to the skin, regardless of the temperature or the duration of application. There was a limited ability to do this over the chest area, provided this was heavily covered with hair; this may be dependent on the hygroscopic qualities of hair.

There was no heightened sensibility in patients with rheumatism or arthritis.

The mucous membranes of the mouth and nose are very sensitive to changes in moisture, and it is possible that our appreciation of moisture in the surrounding atmosphere is elicited through this medium.

The article is to be continued.

WOLTMAN, Rochester, Minn.

A SPECIAL TYPE OF GALVANIC HYPERIRRITABILITY OCCURRING IN "FRAGILITAS OSSIUM." G. C. BOLTON, Monatschr. f. Psychiat. u. Neurol. **49**:144 (March) 1921.

The association of bluish discoloration of the sclerae and abnormal fragility of the skeletal bones, has been repeatedly noted. Burrows states that in fragilitas ossium the sclerae are always blue, but that conversely, blue sclerae may occur independently of abnormal bone fragility. These points have been confirmed by the author's material.

The author reports four new cases of fragilitas ossium, all having certain well defined features: abnormal fragility of the bones; blue sclerae; calcium

deficiency in the long bones; vasomotortrophic disturbances, such as acrocyanosis, chilblains, brittle spotted nails, enamel defects, deficient hair, falling hair, urticaria, migraine, angioneurotic edema, etc.; a special type of galvanic hyperirritability.

The existence of galvanic hyperirritability deserves special note, both because it has not been previously reported in the literature, and because it gives a clue to the genesis of the entire syndrome. Since the studies of Erb we have become familiar with galvanic hyperirritability in tetany. In tetany we find a general lowering of minimal values for all phases of the contraction formula.

In fragilitas ossium, the author found a galvanic hyperirritability of somewhat different type. Values for the closing contractions were little (in some cases not at all) reduced, whereas the values for the opening contractions were always much below normal—never over 3.2 milliamperes. The most marked lowering occurred in the cathode opening contraction (2.8-3.2 milliamperes in this series as against a normal of 6-7 milliamperes). The character of the contraction was normal throughout.

AUTHOR'S TABLE

	Case 1 Ma.	Case 2 Ma.	Case 3 Ma.	Case 4 Ma.	Normal Ma.
CCC	1.2	1.0	0.8	0.8	0.8-1.5
ACC	2.0	2.0	2.2	2.4	2.5-3.0
AOC	3.0	2.4	2.8	3.0	3.0-3.5
COC	2.2	2.8	3.0	3.0	6.0-7.0

A galvanic hyperirritability of similar type has been described by Klose and Vogt in puppies following thymus extirpation. The following table records the type of their findings:

Median Nerve	CCC Ma.	ACC Ma.	AOC Ma.	COC Ma.
8-day old puppy	1.8	2.0	4.2	6.3
Same puppy ten days after thymectomy	1.5	1.5	2.5	2.8

The striking similarity between the galvanic values in his four cases and in thymectomized puppies suggests to the author that in fragilitas ossium, thymus insufficiency plays an important rôle. This seems logical enough. Bone growth is governed largely by three organs: the thyroid, parathyroid and thymus glands. In the author's cases there was little evidence of thyroid insufficiency. The type of bone disturbance, as shown by the roentgen ray, was largely a calcium deficiency. Both the parathyroid and thymus glands play an important rôle in bone calcification. In the cachexia which follows thymectomy, Klose and Vogt find as constant features certain definite bony changes: marked growth inhibition, softness, pliability and even fragility of the skeletal bones; the long bones are brittle and easily broken; spontaneous fractures are common. This indicates that following thymectomy, calcium metabolism is grossly disturbed. Microscopically the calcification zone of the original cartilage is smaller than normal. These changes, which produce a picture of bone atrophy, are, according to Klose and Vogt, the expression of a specific thy-

mectogenic bone disease, and depends on a lack of undissolved calcium, which in experimental animals following thymectomy may be reduced to half the normal.

The author believes that in his four cases we are justified in assuming the existence of a trophoneurosis, that is, a group of metabolic disturbances due to insufficiency of thymus and parathyroids, along with a slight hypothyroidism. This glandular insufficiency rests probably on a hereditary basis. As a necessary result of the diminished function of these sympathicotonic glands, a slight vasomotor insufficiency results.

In Summary: The fragility of the skeletal bones, the blue color of the sclerae, the calcium poverty of the bones, the vasomotor trophic disturbances and the galvanic hyperirritability may be considered as genetically equivalent, or at least closely related. They are all subheads of a trophoneurosis, in which disturbances in calcium metabolism play an important rôle, and which rest on a thymus and parathyroid (perhaps also a slight thyroid) insufficiency.

In view of the infrequency of the condition, one of the case reports is appended.

I. B., a girl, aged 15, whose family history was negative, and who had no neuropathic heredity, had always been nervous. She had had rachitis as a child. Her skeletal bones were abnormally breakable. She broke one leg sixteen times, fifteen times following a slight fall on level ground. Formerly she had gastro-intestinal disturbances, such as gastric pain with vomiting, anorexia and apparently "urticaria interna"; periodic attacks of severe abdominal pain with distention and, for a day or two, diarrhea; this would be repeated after six or eight weeks, independent of errors of diet or of constipation. She had vasomotor trophic phenomena; she frequently suffered from cold hands and feet; in winter she always had chilblains on her hands and feet; her nails were brittle, broke easily and were spotted; her teeth were very bad with a large amount of caries; her upper lateral incisors were much too small; there were some enamel defects; her hair was thin and had a tendency to fall out; cataract was not present. She had paresthesias in the feet (not in the hands) during the winter and often complained of dead fingers (local syncope); sometimes her skin itched. She often had headache, and formerly had hyperacidity and nausea. Her sclerae showed a fairly distinct blue discoloration. The Chvostek phenomenon was present in slight degree. Her pulse was regular and equal with a rate from 82 to 90; her blood pressure was a little low. Blood count revealed, eosinophilia, 6 per cent.; polymorphonuclears, 35 per cent.; large mononuclears, 3 per cent.; lymphocytes, 55 per cent., and transitionals, 1 per cent. The Aschner and Tshermak phenomena were negative. Moderate dermatographia was present. The temperature was normal. Galvanic values for the facial: cathodal closing contraction, 1.5 ma.; anodal closing contraction, 2 ma.; anodal opening contraction, 3 ma.; cathodal opening contraction, 3.2 ma. tion, 3.2 ma.

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BRADYKINETIC ANALYSIS OF SOMATIC MOTOR DISTURBANCES. S. PHILIP GOODHART and FREDERICK TILNEY, Neurol Bull. **3**: No. 9-10 (Sept.-Oct.) 1921.

In this paper the authors give the results of their study of abnormal involuntary movements by means of ultra-rapid moving pictures. They employed the Novograph camera which takes pictures at the rate of 160 to 300 exposures a second (the ordinary cinematographic rate is sixteen a second). The normal

rate of projection on the screen of the rapid moving pictures slows down the movements to such an extent that it permits analysis of every component. This method the authors call bradykinetic analysis.

Although employing the various terms now commonly used to designate the types of abnormal movements, the authors feel that they are inadequate and would therefore substitute descriptive terms. They offer the following objective analysis of adventitious motor elements which can be observed in various conditions:

1. Regular rhythmical oscillations of parts of the body during muscular inactivity or in volitional action.
2. Irregular arrhythmical oscillations of parts of the body during volitional motion.
3. Sudden isolated twitches of the muscles producing involuntary movements or deflecting the course of volitional motion.
4. Sudden successive twitches of muscles producing involuntary movements or deflecting the course of volitional motion.
5. Sustained mobile spasms of muscles during volitional motion producing rhythmical distortions of the limb, trunk or limited parts of the body during activity of the particular part.
6. Sustained tonic spasms of limited groups of muscles during volitional motion which retard or deform the resulting movements.
7. Sustained tonic spasm in a large number of muscle groups during activity or in rest, producing distortion of the body together with marked deformity of motion during action.

The first case studied was one of chronic degenerative chorea in a woman. The normal rate of projection showed irregular, meaningless, purposeless movements. Bradykinetic analysis showed that these movements apparently had a definite associative significance. They comprised a series of symbolic attitudes indicative of emotion and expressing entreaty, or a negative self-feeling of shame. These associated movements and emotional expressions would link the disease with the corpus striatum and optic thalamus and also point to the subconscious controlling level in hysteria. The analysis also brought out the failure of the kinetic mechanism controlling associated movements and impairment of the static mechanism.

The second case was one of chronic degenerative chorea in a man, with some elements of dystonia. Here analysis also brought out attitudinizing, but of the positive feeling tone characteristic of the male. There were, besides the choreic movements, sustained tonic spasms producing distortions which are manifestations of hyperkinetic innervation of muscles recognized as the essential part of the syndrome of dystonia.

Analysis of the third case, which was one of multiple sclerosis, showed a disintegration of the synergic units throughout the body, an incoordination between agonists and antagonists, a failure in the check elements of the opposing groups of muscles. The tremor was shown to be the result of a static defect, due to the dissociation of the synergic units of the body, consequent on the disproportionate innervation of the dominant and check elements in the muscle groups. This proves that the "tremor" is cerebellar in origin.

Case 4 was diagnosticated clinically as a functional astasia abasia with excessive isolated and successive spasms of the muscles of locomotion. Analysis showed that these movements were expressive of acutely conscious and psychically much magnified fear, in contrast with the simpler attitudinizing of

chorea. A wavelike contraction in the muscles, possibly significant of functional dyskinesis, was also observed.

Study of three cases of dystonia musculorum deformans in a brother and two sisters revealed disturbances of movement due to a partial failure of coordinate time reaction in groups of agonists and antagonists. There was excessive innervation of both the kinetic and static elements of motion with the resultant extreme degree of movement and posture, in many instances amounting to actual distortions. This marked overflow of innervation was apparent in nearly every group of muscles in the body, producing needless and often untimely postures as well as active movements wholly unnecessary to the acts performed.

Analysis of another case of dystonia with extreme sustained spasms again showed an unchecked overflow of kinetic energy and a static dissociation which threw the patient into involuntary attitudes. Study of two other cases of dystonia, one with sudden ticlike movements, "dromedary attitude" and semilunar foot, further demonstrated the disorganization between the static and kinetic mechanisms. All patients with dystonia showed the imbalance in the ratio of the two kinds of energy, especially during volitional effort. The disturbance in the rhythm of the two mechanisms, due to improper synchronization in time, force and rate of action, leads to a conflict between the hyperstatic and hyperkinetic innervation.

The last case to be analyzed was one of cerebellar ataxia due to cerebellar aplasia. The ataxia or asynergia was shown to be due to failure of proper coordination in the synergic units of the body and to disproportionate innervation of the dominant and check elements of the individual units.

The authors came to these conclusions:

1. Bradykinetic analysis permits minute study of disturbances of motion.

2. Adventitious elements in motor disturbances are manifest as disorders of the kinetic or static mechanism.

3. All purposive movements depend on the concurrent and synchronized operation of the kinetic and static mechanisms. Such movements are made up of a series of postures.

4. The kinetic mechanism is represented by a neural mechanism capable of correlating the impulses necessary to the production of purposive movements, attitudes and postures.

5. The static mechanism is represented by a neural mechanism capable of correlating the impulses necessary to the maintenance of postures.

6. Present evidence warrants the allocation of the chief center of the static area in the cerebellum and the kinetic in the end brain, with the primordial center of the second in the striatum.

7. The disorders of motion investigated may be attributed to disorganization in the two fundamental neural areas.

8. Choreiform movements of degenerative chorea are due to a hyperkinetic disorder resulting in the sudden production of postures and attitudes expressive of emotions. In astasia abasia the movements the emotions express are of a more complex kind, indicative of psychic elaboration.

9. Cerebellar ataxia and intention tremor are due to the same defect of the static mechanism-disorganization of the synergic units.

10. Dystonias are both hyperstatic and hyperkinetic in nature, with the absence of normal concurrence between the two streams of innervation. These mechanisms appear to be affected by pronounced irritation or lack of proper inhibition.

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EXPERIMENTS ON THE TRANSPLANTATION OF LIMBS IN AMBLYSTOMA. FURTHER OBSERVATIONS ON PERIPHERAL NERVE CONNECTIONS. S. R. DETWILER, J. Exper. Zoology **35**:115, 1922.

Shifting the position of the fore-limb rudiment of Amblystoma embryos in the tail bud state a given number of body segments anterior or posterior to its normal site does not effect to the same extent a corresponding shifting of the segmental nerve contribution to the brachial plexus. There is a marked tendency for transplanted limbs to receive innervation from the normal limb level of the cord. The number of segments occupied by a transplanted limb does not determine the number of the segmental nerves contributing to its plexus. When the fore-limb rudiment is transplanted from two to three segments anterior to the normal position, the distal portions of the normal limb nerves are found to grow anteriorly to the distance of several segments to effect functional connection with the heterotopic appendage. The proximal portions of the nerve follow a normal pathway. In effecting connections with the limb, the nerves apparently grow anteriorly against the mechanical opposition of the developing myotomes, which tends to direct them caudally.

The results of the present limb experiments strongly suggest that there exists between the limb and its normal nerves a developmental relationship which is more intimate in character than any developmental association between these same nerves and other structures. The contention is thus supported that mechanical influences, although governing in large measure the character of nerve pathways, do not reveal the mechanism by which proper connections are made at the periphery. It is probable that there exists at the periphery forces of a chemotactic or galvanotactic nature which bring about orderly selectivity.

The function of transplanted limbs is conditioned by four main factors: (1) structural incompleteness of the shoulder girdle, (2) defective development of the limb and the shoulder muscles, (3) defective peripheral innervation, and (4) defective connections within the central nervous system. The gradual loss of function in limbs as they are transplanted farther and farther away from the normal situation is attributed to increased defective connections with the central reflex pathways involved in normal limb locomotion, or rather to a corresponding decrease in effective peripheral innervation and structural deficiency of the shoulder and limb. The high percentage of abnormalities in limbs transplanted from two to three segments anterior to the normal position is due to the fact that they are placed in the environment of active gill-forming tissue. In these cases the gill-producing properties of the tissue surrounding the transplant markedly disturb the normal posture and differentiation of the appendage. When the ectoderm and mesoderm are removed from the entire external gill swelling and a limb is transplanted into the denuded area, abortive gills may develop from the surrounding tissue. The results of the experiments indicate that the tissue lying ventral to the typical gill region possesses a relatively higher gill-forming capacity than does that lying anterior or dorsal. That the outgrowth factors for the external gills reside not only in the ectoderm of the immediate gill region, but extend with diminishing degree of intensity into the surrounding ectoderm, is evident. The hypoglossal nerve in Amblystoma is formed by the union of the ventral rami of the first and second spinal nerves and corresponds with the arrangement characteristic for urodeles. The entire ventral ramus of the second spinal nerve may contribute nerves to the transplanted limb. In such cases the hypobranchial musculature is supplied by the first spinal nerve. When the ventral portion of the first myotome

is excised with the gill mass, the anterior segments of the sternohyoid muscle, the geniohyoid muscle and the hypoglossus muscle are absent on the operated side. The hypoglossal trunk in such cases does not assume its normal anterior pathway, but terminates in the posterior segments of the sternohyoid muscle.

WYMAN, Cambridge, Mass.

DISTURBANCES IN RELATION TO COLORS IN APHASIC PATIENTS.

OTTO SITTIG, Monatschr. f. Psychiat. u. Neurol. **49**:63 (Feb.) 1921, and **49**:159 (March) 1921.

Color blindness may be congenital or acquired. The acquired type may be peripheral or central in origin. The brain pathologist is chiefly interested in acquired color blindness of cerebral origin.

In 1884, Wilbrand described a group of cases in which the patient is able to recognize and distinguish all colors but is unable to associate the color with its appropriate name; that is, he misnames colors which are shown him; if a color is named, he cannot pick it out of a group; and he is unable to name the color of familiar objects. This disturbance though bearing a superficial resemblance to color blindness, is in reality an aphasia. Wilbrand called it amnestic color blindness.

PATIENTS' BEHAVIOR TOWARD COLORS

Case	Sorting of Colors	Naming of Colors	Understanding of Names of Colors	From Memory: Colors of Objects		Color Shown, Name an Object of This Color	Appreciation of Falsely Colored Pictures
				Naming	Showing		
1	Disturbed	Disturbed	Disturbed	Disturbed	Disturbed	Disturbed	Usually good
2	Disturbed	Disturbed	Disturbed	Good	Disturbed	Disturbed	Good
3	Disturbed	Disturbed	Disturbed	Disturbed	Partly disturbed	Disturbed	

The author describes three cases:

CASE 1: Gunshot wound of the left occipital region, with injury to the underlying brain tissue. The patient showed the remains of a temporal lobe aphasia, with difficulty of word finding as the outstanding feature.

CASE 2: Thrombotic softening on an arteriosclerotic basis. Clinically this patient showed a pure alexia and a word amnesia.

CASE 3: Tumor which had led to temporal lobe aphasia with marked disturbance of word finding.

The chief interest in these cases centers in the behavior of the patients toward colors. An analysis of this behavior is found in the table.

In all three cases accurate tests showed that color perception itself was normal. Referring to the table, we find that the three cases have certain things in common. The patient cannot correctly name colors; he has lost understanding of the names of colors. If an object is named to him (lemon, cherry) he is unable to name the color of that object. In explanation of these phenomena we are justified in assuming a pure aphasia for names of colors—"Farbennamenamnesie," in Wilbrand's sense. But this is not the whole story; when an object is named to the patient, the color of that object cannot be picked out of a group of colors. There have been several attempts to explain this. On purely theoretical grounds, there are three possibilities.

1. Disturbance of recognition (Erkennen). Color perception per se is normal, as demonstrated by appropriate tests. But this does not mean that color recognition is normal. We know in other fields that perception may be intact, and yet recognition be faulty, due to disturbance of the higher psychic functions. Such disturbances are classified as agnosias. The author calls attention to a possible analogy to mind blindness, and considers the possibility of a classification of this disturbance as a mind-color-blindness. Positive proof of this is difficult.

A second possibility of which much has been made is a disturbance of color memory. Lewandowsky supported this view; others have opposed it.

The third possibility is to consider this inability to pick out of a group of colors the color of an object named an integral part of the aphasic disturbance. Unquestionably the knowledge of the names of colors has some influence on color recognition; but the author is convinced that this alone is insufficient, and that other factors must be sought.

Still more difficult to explain is the disturbance in sorting the Holmgren test colors. This was striking in all of the author's cases, and at first suggested a color blindness of perceptual origin. But careful tests with the Helmholtz color mixing apparatus showed that color perception per se was normal. In analyzing this, the author points out that the Holmgren test is not simply a matter of matching colors. If one changed the Holmgren test about and asked the patient simply to give the exact match for the test color, it worked very well. It was only when all the shades of a given color were to be grouped that difficulties arose. Evidently the trouble lay in abstracting the shade, and bringing the different individual perceptions into a higher concept. Here we are dealing with higher psychic processes of abstraction, attention, evocation of the color concept and grouping of essentials in addition to simple perception.

Summing up the various features of his cases, as shown in the table, the author concludes that the sum total of the phenomena are dependent on the one hand on aphasic disturbances (inability to name colors, etc.) and on the other hand on disturbances of the higher psychic functions (failure to pick from a group, the color of an object named; failure to sort the Holmgren test colors). He calls attention to certain analogies to the explanation of alexia as given by Kramer, and von Stauffenberg. Kramer considers alexia as a summation of slight optic disturbances, plus slight speech disturbances. Von Stauffenberg speaks of alexia as a combination of optic agnosia and speech disturbance. In considering the various color phenomena which his patients have presented the author feels that here, as in alexia, the best explanation is to be found in a summation of speech disturbances and optic agnosia. It is noteworthy that in all the cases of the group either mind-blindness, or alexia, or aphasic troubles (especially amnestic aphasia) were found.

As to the localization of the process, the phenomena described occur in lesions of the posterior portions of the brain, especially in the parieto-occipital lobes.

Summary: 1. We must differentiate between true color blindness and disturbances in relation to colors in which color perception is intact.

2. The latter are characterized by the inability of the patient to name colors correctly, or to understand the names of colors. This is an aphasic disturbance and may be called amnesia for names of colors (Farbennamenamnesie).

3. There are cases in which in addition, color sorting is disturbed. Here the assumption of an aphasia alone is not sufficient. We must assume agnostic disturbances also.

4. These disturbances are often associated with alexia or mind blindness or amnestic aphasia.

5. One hemisphere (usually the left) is dominant.

6. This disturbance in relation to color is complicated and built up of several component parts, which influence each other in such a way that separation of the individual factors is difficult. Analogous to Kramer's and von Stauffenberg's explanation, it is a summation of aphasic and optic-agnostic factors. In some cases the aphasic, in others the agnostic factors predominate. The agnostic factors are to be considered as disturbances of the higher psychic functions as attention, evocation of concept, etc.

7. Owing to the complexity of the phenomena, much further work is required.

8. On anatomic and clinical grounds, one may assume that the lesion is in the parieto-occipital zone.

SELLING, Portland, Ore.

DEMENTIA PRAECOX AND THE ADOLESCENT PSYCHOSES FROM THE ORGANIC VISCERAL VIEWPOINT AS REGARDS PROGNOSES AND THERAPY. DANIEL J. MCCARTHY, Am. J. Psychiat. **1**: No. 3 (Jan.) 1922.

McCarthy presents an interesting diagnostic-therapeutic discussion of the adolescent psychoses. They are not synonymous with dementia praecox, and although the reaction is often schizophrenic, one must regard them merely as borderline psychoses. Whether or not they would have progressed to classical syndromes cannot be determined as the great majority are readjusted by appropriate therapeutic endeavors. The nondegenerative group may be sub-divided into: "(*a*) Those whose toxic factors were dependent on pulmonary disease, at the first development period, leading to constitutional and endocrine disturbances at the adolescent period. (*b*) The syphilitic group when early acquired, or inherited syphilis produces a similar reaction as in (*a*). (*c*) Steadily progressive infections in the early adolescent period, belonging in a general way to the focal infections, and leading to reactionary states in the endocrine field. (*d*) Nondetermined infections with thyroidal disturbances. (*e*) Stress reactions in the moron groups, due either to infections or overload leading to a praecox syndrome."

The author believes that many dementia praecox symptoms may be found in embryo in normal adolescence and that a psychologic balance is attained without outside assistance. There is a definite pathologic group that may be classified into: "(*a*) Those whose symptoms are referable to introspection concerning some personal physical characteristic. (*b*) Those in whom problems of a neurosexual nature are the disturbing factors. (*c*) External problems in relation to family affairs leading to a feeling of instability in reference to life and other matters. (*d*) Visceral disease, with an attempt at adjustment for insufficiency, either in purely social matters, at school or in business affairs." The percentage of cases in these groups developing into serious problems depends essentially on the intensity and effect of fundamental instability and continuing intoxication. If there is no or only a slight degree of instability the intoxication must be extremely severe, or if there is gross lack of fundamental equilibrium, the toxic factor may be mild in degree and yet produce a deleterious mental result.

McCarthy's prognostic optimism springs from his aggressive therapeutic tactics. He is not primarily concerned with the particular type of insanity, but with a careful and accurate diagnostic estimate of: "(*a*) The physical machine

as a whole. (*b*) Disease and physical damage of organs. (*c*) Functional derangement of viscera. (*d*) An estimate of the effect of these on the nutrition of the brain through damage to its nutritional medium (the blood) by alteration in its content (anemia, etc.), or by the addition of toxic material from one or more organs. (*e*) Psychogenic factors. (*f*) Mental symptoms." The mode of treatment is strictly individualized and takes its keynote from the carefully accumulated fund of diagnostic information. In addition, there are certain measures which find free application. They include rest, fresh air, postural and gymnastic therapy, occupation, hydrotherapy, discipline, etc. They are given with minute and unswerving attention to detail. Under such a diagnostic therapeutic regimen 80 per cent. of the patients with adolescent psychoses recover.

STRECKER, Philadelphia.

INTERMITTENT CLAUDICATION. H. SCHLESINGER, Med. Klin. **17**:1507 (Dec. 11) 1921.

Intermittent claudication is characterized by pains, muscle cramps, severe paresthesias, limping and even profuse perspiration occurring in the lower limbs on walking, subsiding rapidly with rest but again precipitated when walking is resumed. Arterial pulsation in the affected limbs is usually impaired, and vessel changes are as a rule demonstrable.

The diagnosis, while relatively simple, is frequently missed because of an inadequate history and the failure to palpate the arteries in the feet.

Persistent neuralgias, ischalgias, meralgia paresthetica, tarsalgia, and flatfoot should always suggest an examination of the pedal arteries. Muscular rheumatism and hip joint diseases are not infrequently confused with a claudication.

Erb was the first to postulate an arterial change as the etiology of intermittent claudication. Nicotin, exposure, diet and nervous influences are mentioned as factors in the production of this disturbance. Impaired venous circulation and thrombus formation are occasionally found, but as a rule varicosities do not play a part in the picture.

It is not uncommon to find associated with claudication a neuritis which does not disappear on rest. The neuritis is apt to be unilateral accompanied by loss of Achilles and patellar reflexes and some sensory changes. The neuritis usually clears up with improvement in the circulation.

Vasomotor phenomena are frequently present and show themselves in various ways. There may be arterial contraction, with pallor of the affected limb and decrease in temperature. This may persist even when the limb is placed in the horizontal position. Occasionally in place of the arterial contracture a dilatation occurs. A rather unusual phenomena is the production of blanching in the affected limb by local heat. Vasodilator drugs tend to cause a reaction in the diseased limb in preference to the sound one. Thus an injection of sodium nitrite will produce redness and increased warmth in the affected limb for several hours.

The prognosis, according to the author, is not entirely hopeless. A rigid prophylaxis is of the utmost importance. Exertion and exposure should be avoided. Smoking should be discontinued as these people seem to have an idiosyncrasy for nicotin. In spite of the usually accepted opinion that heat is beneficial, the author believes that more harm than good results from it. Moderate heat may be employed. In the protection from cold maceration must be avoided. Galvanization, as recommended by Erb, has produced good results.

Internal medication in the form of theobromin, aspirin and nitroglycerin is recommended. Sodium nitrite if used must be given subcutaneously. The latter method, in the hands of the author, has been most successful. Strychnin has also been employed. Wieting's operation—the planting of the femoral artery into the femoral vein—is not to be recommended.

In the author's experience the results are much better than one might suppose from a review of the literature. Long remissions with return of function and ability to resume work are not uncommon.

MOERSCH, Rochester, Minn.

THE EFFECT OF TRANSPLANTING A PORTION OF THE NEURAL TUBE OF AMBLYSTOMA TO A POSITION AT RIGHT ANGLES TO THE NORMAL. H. L. WIEMAN, J. Exper. Zoology **35**:163, 1922.

When a small section of the neural tube of Amblystoma (equal to the length of two somites), at the stage of the closed neural folds, is removed at the level of the second to fourth somites, together with portions of adjacent somites, and reimplanted at right angles to its normal position, the transplanted tube continues its development and retains its original polarity. A preliminary dorsal flexure of the body occurs, owing to contraction accompanying the closure of the wound, followed by straightening and a ventral flexure. When the notochord was cut and moved as a part of the transplant, the ventral bending was practically absent. Another general effect of the operation was the underdevelopment of the gills. The embryos were tested for nervous conductivity by delicate tactile stimuli.

The growing brain, pushing the anterior stump back against the anterior side of the transplanted tube, may be a factor in forming a union at the point of contact. Nerve fibers grow back from the anterior stump into the transplant near its original anterior end. The posterior stump becomes club-shaped but shows no initial tendency to send fibers forward into the transplant. A connection between the transplant and the posterior stump is brought about by the continued growth backward of fibers from the anterior stump through the transplant. Ascending fibers then grow forward from the posterior stump. The transplant eventually becomes absorbed in the reconstructed neural tube, but the appearance of sections shows that the cells of the transplant do not lose their original polarity as late as thirty to forty days after operation. The results point to the formation of the long motor tracts from the brain to the cord before the sensory tracts. The restoration of nervous connections through the transplant takes place in the direction of the normal metabolic gradient of the embryo.

When the operation is performed at a stage just before the larvae become sensitive, the process is the same, but the union is formed in a shorter time after operation. When the operation is performed at the earlier stages, but in the region of the fifth and sixth somites, or farther back, a longer time is required for the formation of nervous connections. In a series of twelve operations no case of complete connections between the stumps occurred in thirty days. In one case at least the transplant was disintegrated at the end of thirty days. If the initial step in the restoration process is a posterior growth of fibers from the brain to the transplanted tube, the longer time required for fibers to reach the more posterior level would allow somites transplanted with the tube to develop to such an extent as to delay or prevent the formation of a nervous connection. The nearer to the brain the transplantation is made the better the chances for connections to be established.

WYMAN, Cambridge, Mass.

AN UNUSUALLY ACUTE CASE OF MULTIPLE SCLEROSIS WITH ANATOMIC FINDINGS. Von Weizäker, Monatschr. f. Psychiat. u. Neurol. **49**:221 (April) 1921.

It is very difficult to make a differential diagnosis between certain cases of multiple sclerosis and the encephalitides and myelitides. The line of demarcation between the two seems constantly to be growing more indefinite; this is true on the anatomic as well as on the clinical side. There is an increasing tendency to assume an inflammatory origin for multiple sclerosis. Confirmation of this view would explain why one finds all transitions between the two types of diseases.

A patient aged 57, had syphilis at the age of 17. He received six courses of treatment. Eight years ago he had diplopia of short duration. The present illness began eight days before admission to the hospital, with headache, weakness, instability of gait, weakness of the hands and a dead feeling in the little finger. Then diplopia and hiccup began. On admission he had fever, cyanosis and stupor. The pupils were equal, reaction to light slow, convergence reaction normal. He had moderate ataxia which was increased on closing the eyes. The spinal fluid findings were: globulin one plus, cell count 20, Wassermann test (0.2-1 c.c.) negative. The blood Wassermann test was also negative.

The patient had fever and perspired. The reflexes remained normal. There was a certain rigidity of the muscles, and the face was somewhat masklike. The stupor gradually deepened. Death occurred fourteen days after the first prodromes, ten days after the actual onset of symptoms.

At necropsy there was no evidence of syphilis. In the white matter of the cerebrum, pons and cerebellum were a few foci which differed in no respect from the plaques of a chronic multiple sclerosis. In addition, there was an acute process, most marked in the pons and medulla, but extending into the upper cord. The acute changes were most extensive in the gray matter, but also involved the white matter. They included definite ganglion cell degeneration with neuronophagia and a marked glia reaction. There was a general reaction of the vascular connective tissue with small round cell infiltration throughout the vessel walls.

The anatomic and the clinical picture both showed an acute process superimposed on a chronic. Unquestionably the plaques were plaques of multiple sclerosis. The difficulty arises in the interpretation of the acute changes. These were changes which one might find either in an acute multiple sclerosis or in an acute encephalitis, especially a lethargic encephalitis. How shall we interpret them?

The author feels justified in considering the case one of multiple sclerosis because of the typical plaques in the white substance with sparing of the cortex, with special involvement of the medullary sheaths, and with marked productive glia reaction. If we accept this view, and do not assume two separate diseases, this case supports the views of those who, with Marburg, Anton and Wohlwill, Jacob and others, believe that the acute patch of multiple sclerosis differs in no respect from the lesions of such definitely exogenic diseases as myelitis, poliomyelitis and lethargic encephalitis.

In this case the acute phenomena dominate the picture; the typical chronic plaques are subordinate. Clinically the only sign pointing to the possible development of a multiple sclerosis was the diplopia which was present eight years ago.

As long as we cannot demonstrate the causative agent, we cannot give a positive answer to the question. The author favors the grouping of this case as a multiple sclerosis.

Selling, Portland, Ore.

STUDIES ON SPATHIDIUM SPATHULA. I. THE STRUCTURE AND BEHAVIOR OF SPATHIDIUM, WITH SPECIAL REFERENCE TO THE CAPTURE AND INGESTION OF ITS PREY.

LORANDE LOSS WOODRUFF and HOPE SPENCER, J. Exper. Zoology **35**:189, 1922.

Pedigreed cultures of Spathidium were kept in moist chambers and fed on beef extract plus hay infusion seeded with small ciliates, chiefly Colpidium. The typical vegetative form is flask-shaped, but encystment, division, conjugation and starvation alter the typical form. There are sixteen longitudinal, double rows of cilia evenly distributed over the cell. At the edge of the truncated anterior end there are two rows of longer, stouter, cilia. The cytostome is a long, narrow depression extending nearly the length of the truncated end at right angles to the axis of the cell. Spathidium does not possess trichocysts, and consequently the extrusion of such structures is not responsible for the paralysis of the prey. Trichites are present in the oral region and form a paling which is apparently comparable with the supporting rods about the mouth in the more generalized gymnostomatous ciliates. There is no conclusive evidence that the trichites are the locus of the poison. There is one large contractile vacuole at the posterior end of the cell. In the typical vegetative cell are two long, ribbon-like macronuclei which extend nearly the whole length of the cell. There are from six to nine micronuclei. Therefore the Spathidium studied by Moody must be interpreted as an amicronucleate race.

The food of Spathidium is limited to small ciliates but not to members of the genus Colpidium. Spathidium swims forward quite rapidly, revolving on its long axis. In general its behavior can be interpreted in accordance with the "avoiding reaction" as worked out by Jennings. When a swimming Spathidium is stimulated it reverses its cilia, moves back a little and then proceeds in a slightly different course. When the anterior end strikes a small ciliate the prey becomes motionless and exhibits pathologic changes. If the prey is instantly paralyzed and remains against the oral region of its captor, the Spathidium gives a few rapidly repeated avoiding reactions, the prey is encircled by the cytostome and sinks into the endoplasm. Ingestion takes place in about thirty seconds. If the prey is not instantly paralyzed and moves a short distance away, the behavior of the Spathidium is not identical in any two cases. In general the Spathidium gives a series of avoiding reactions until sooner or later its mouth is in contact with the food. The reactions are modified and complex, indicating "sensing at a distance." The factor involved in this "sensing" is apparently a chemical substance secreted by the Spathidium when the prey is paralyzed, because the prey is a center of attraction for other Spathidia in the neighborhood, and the oral region of the Spathidium in question is also a stimulus to neighboring specimens. Spathidium comes in contact with its prey solely by chance.

WYMAN, Cambridge, Mass.

SYPHILIS AND TRAUMA: THE WORKMEN'S COMPENSATION ACT, THE INDUSTRIAL PHYSICIAN AND THE SYPHILITIC EMPLOYEE.

JOSEPH V. KLAUDER, J. A. M. A. **78**:1029 (April 8) 1922.

A latent syphilitic infection in some cases may play an important rôle as a factor in the immediate and late consequences of trauma. The matter of compensation in cases of injury to workmen and syphilitic soldiers during the recent war has made the question of the relationship of trauma to syphilitic lesions a most important one. It is known that trauma may determine the

site of tuberculous lesions, particularly of the bones and joints, and the same is true for other conditions, such as psoriasis; in reference to syphilis the relationship to trauma has been noted in relationship to chancre and to the mucous patches of syphilitic glossitis in excessive smokers. Spirochetes from the blood may be deposited at the traumatized site. The phenomenon allergy, or the hypersensitive state of syphilized tissue, doubtless plays a rôle in the influence that trauma exerts in causing syphilitic lesions. Because of the allergic state of syphilized tissue, it is perhaps possible that trauma, a nonspecific irritant, acts as a stimulant to this tissue, causing it to react in a characteristic manner in the absence of the specific irritant, namely, *Spirochaeta pallida.*

Dr. Klauder says that the fact that the workman had a preexisting disease —syphilis—does not alleviate the responsibility of his employer for compensation for injury, disability or death indirectly due to syphilis. The fact of trauma being able to influence the course of a patient with syphilis makes the examination of industrial workers one of great importance. An examination of the spinal fluid of all syphilitic workmen is, of course, impossible, but the most important and perhaps the first objective sign of neurosyphilis is pupillary abnormality, and while these changes are not an infallible symptom of neurosyphilis, they are suggestive. The author believes that a syphilitic workman with pupillary abnormalities on the clinical side, and a positive spinal fluid on the laboratory side, is a potential candidate for paresis if he is retained in a position of a hazardous nature in which he may sustain trauma. Such an employee should be given other work.

NIXON, Minneapolis.

CONSTRUCTIVE FORMULATION OF SCHIZOPHRENIA. ADOLF MEYER, Am. J. Psychiat. **1**:355 (Jan.) 1922.

Under this title Dr. Meyer calls our attention to the "potentially modifiable points" of dementia praecox reactions. He is interested, as are all who have a psychatric bent, in the manic-depressive patient who becomes demented and in the dementia praecox patient who recovers. There is nothing so worthy of consideration as the schizophrenic process; occasionally a physician with a certain understanding of this process helps a patient through a crisis which means life or death.

In describing his method of studying a patient, Dr. Meyer uses some terms not frequently used. In a given case he would rule out the organic brain diseases and the infective-toxic reactions; he would then differentiate the psychoneuroses from the wide group of more functional disorders which concern the whole person and include exhilarations, depressions, schizophrenic reactions and transition forms. In the latter group, he would make a close study of the affect and set such impure manifestations as fear and tension against the pure elations and depressions. The content must also be examined to determine how well it fits in with the affect; if it does not fit in and cannot be explained by the influence of toxins or infections, the patient probably has schizophrenia which is an "ominous," but not a hopeless condition. Each patient's response to efforts of adjustment must be tested, and in that test lies considerable hope. Certainly every one working with mental patients should be familiar with this point of view and understand the claim that "terminology is an absolute secondary issue" while the "summary formulation of the facts is primary."

In a paragraph Dr. Meyer calls attention to the need of study of the unmeasured metabolic symptoms, which include unaccountable fever, refusal of food and incongruous pupil reactions, which are so prominent in patients with mental disease that it is remarkable that they have been neglected.

BOND, Philadelphia.

THE TREATMENT OF MULTIPLE SCLEROSIS. CHARLES METCALF BYRNES, J. A. M. A. **78**:867 (March 25) 1922.

The author emphasizes the fact that care must be exercised in the differentiation of lesions of multiple sclerosis as there is a striking analogy in the symptomatology and pathology of insular sclerosis and certain types of cerebrospinal syphilis. The lesion in multiple sclerosis in all probability is of an inflammatory nature. There are cellular reactions which resemble those of syphilis and, with the exception of the reaction to the Wassermann test, the spinal fluid exhibits changes comparable to those in vascular and gummatous types of cerebrospinal syphilis. He thinks the failure to demonstrate *Spirochaeta pallida* or to secure a positive Wassermann reaction in all cases is no proof that syphilis may not be an etiologic factor. On the other hand, a positive Wassermann reaction in multiple sclerosis does not establish its specific nature, since the two conditions may exist simultaneously or, in such instances, the disease is syphilis and not multiple sclerosis. For this reason the therapeutic test is of no value as a diagnostic measure.

While syphilis may not be an etiologic factor there are undoubtedly clinical types of disseminated syphilis that are not distinguishable from insular sclerosis so that until the etiology of the latter disease is established, or a more successful form of treatment found, the author believes the adoption of antisyphilitic treatment is worthy of consideration, and that the treatment is more likely to be effective if begun early. He recommends both intravenous and intradural medication.

NIXON, Minneapolis.

THE TEMPERATURE SENSES IN THE FROG'S SKIN. ANN HAVEN MORGAN, J. Exper. Zoology **35**:83, 1922.

Receptors for heat and cold were demonstrated in the frog's skin by suspending a reflex frog and dipping the foot into water of various temperatures. The lowest degrees of heat which stimulate the skin lie somewhere between 35 C. (95 F.) and 41 C. (105.8 F.). The first responses to cold occur at 10 C. (50 F.). As the heat is increased the reaction times decrease regularly from long intervals to short ones (25 seconds to 1 second). The interval between stimulation by cold and the response was an inconsiderable period. The typical response to heat is an upward jerk of the leg. The typical response for cold is a rigidity and tenseness of the muscles, but there may be an upward jerk. It is impossible to stimulate the foot of a reflex frog by gradually increasing the temperature without response, the first response occurring at 43 C. (107.4 F.). The reaction times indicate that the heat receptors are in the deeper and the cold receptors in the more superficial layer of the skin. The sense of heat may be affected by treating the foot with cocain solution, and both the sense of heat and that of cold may be wholly eliminated by cutting the sciatic nerve or by removing the skin. Treatment with cocain entirely removes the sense of cold. It has been possible to isolate the temperature sense from the tactile, pain and chemical senses by treating the foot with

cocain solution and stimulating it by dropping shot on it, pricking the toe with a needle or treating the foot with acid. Responses to heat and cold persist beyond response to touch. Pain persists beyond heat and cold. Response to acid (0.5 per cent. hydrochloric) persists beyond response to heat or cold. Heat persists beyond cold. The receptors are well defined.

WYMAN, Cambridge, Mass.

SEQUELS OF ACUTE EPIDEMIC ENCEPHALITIS: A STUDY OF NINETY-TWO CASES FROM ONE TO THREE YEARS AFTER RECOVERY. MORRIS GROSSMAN, J. A. M. A. **78**:959 (April 1) 1922.

In recent months several papers have appeared showing that earlier rather optimistic prognoses in cases of epidemic encephalitis were not altogether justifiable. Further, the seriousness of the paralysis agitans syndrome both from the standpoint of the outlook of those presenting that picture at the beginning and of the possibility of patients who had apparently recovered acquiring this syndrome months after the original attack is being noted.

Of the ninety-two cases reported only ten patients had fully recovered; fourteen were functionally well, two remained stationary and four showed some improvement. Sixty-two of the ninety-two patients showed evidence of a more or less progressive involvement of the central nervous system, and of this group forty-two patients presented a clinical syndrome closely resembling that of paralysis agitans. Nearly 70 per cent. of the parkinsonian group were under 30 years of age and, in contrast to true paralysis agitans, a large number of these patients showed pupillary changes and residual signs pointing to cranial nerve involvement; the facial nerve most frequently presented abnormalities.

The remaining twenty progressive cases showed various types of motor disturbance, such as choreiform and choreo-athetoid movements, ticlike movements of the head, torsion spasm of the pelvis and leg, myasthenia gravis and spontaneous tremors.

It is obvious from a study of this group of patients that the danger from epidemic encephalitis does not pass when patients recover from the acute illness.

NIXON, Minneapolis.

THE NATURE OF FUNCTIONAL DISEASE. WILLIAM MCDOUGAL, Am. J Psychiat. **1**:335 (Jan.) 1922.

In this day of insistence on the somatic causes of mental disease, it is interesting to read a paper which insists on the independence of the mind. Dr. McDougal believes that the too persistent search for structural defects has retarded psychiatry and that function will determine structure. If we find a disordered organism (hallucinations) with no structural defect visible, we cannot assume that with better methods we could always find the mechanical defect. The organism differs from a machine because its purpose lies within itself and it is regulated and adjusted from within. He insists on the importance of timing relations. "Paralyses and contractions, which play so large a part in the picture of functional disorders, may properly be regarded as due to . . . breakdown of the power of adjustment of time relations." "Purpose works . . . by timing the spark." The compositor unfortunately makes this determine "the moment of the conversion of potential to lunetic (sic) energy."

Cases of dementia praecox reported by Jung and Forsyth are given to show the apparent onset of the disease under unfavorable mental influences and improvement or recovery on the removal of bad or the adding of better mental factors. The consideration of Motts' pathologic studies leads the writer to suggest that diminution of function may produce the structural changes which are found and to say that such studies do not establish precedence in time. Finally, the writer says, "I believe that the mind has a nature and structure and functions of its own which cannot be fully and adequately defined in terms of structure of the brain and its physical processes." Or, there may be "mental diseases which are of the mind only and not at all of the body."

BOND, Philadelphia.

ANATOMIC STUDY OF INJECTION OF SECOND AND THIRD DIVISIONS OF TRIGEMINAL NERVE. F. C. GRANT, J. A. M. A. **78**:794 (March 18) 1922.

Injections into the second and third divisions of the nervus trigeminus have become a common procedure. They were used primarily almost only for the relief of trigeminal neuralgia; the present indications for blocking these nerves include many dental and surgical manipulations on the face and jaws.

Most of the descriptions of the technic of these injections are disappointing in their practical application. To reach successfully a nerve trunk lying deep beneath the skin and emerging from bony orifices in the skull, definite landmarks and angles are required as guides to the approach. In the clinic with which Dr. Grant is connected there has been developed an instrument called a zygometer which aids in determining accurately the point on the face at which the needle should be introduced to reach a particular nerve trunk; using this instrument to standardize the points of insertion of the needle through the skin, the angles in the horizontal and vertical plane through which the needle must pass from this fixed surface point to enter the nerve trunk have been worked out. Details of the application of this procedure are given.

NIXON, Minneapolis.

THE IMPORTANCE OF ENDOCRINE THERAPY IN COMBINATION WITH MENTAL ANALYSIS IN THE TREATMENT OF CERTAIN CASES OF PERSONALITY DEVIATION. EDITH R. SPAULDING, Am. J. Psychiat. **1**: No. 3 (Jan.) 1922.

Spaulding presents four patients who were brought to a social adjustment by a combination of mental analysis and reeducation plus ductless gland therapy. The diagnoses were manic-depressive psychosis, psychopathic personality, psychoneurosis with antisocial tendencies and emotional imbalance. In three instances there was principally pituitary deficit and in one an overactive thyroid gland. The author deplores the singleness of approach to the individual patient which is largely dictated by the scientific inclination of the particular physician who studies the case. It is emphasized that the need is often twofold, and it is suggested that the psychiatrist enroll the endocrinologist as his chief aide. It might be added that especially in considering the problem of delinquency, the need may be even greater, and that it is often useless to take therapeutic steps until one has clearly in mind and is able to assign a proper place to all the psychogenic and physical liabilities and assets.

STRECKER, Philadelphia.

CLINICO-ANATOMIC STUDY OF A FATAL CASE OF MYASTHENIA (ERB-GOLDFLAM). P. MARIE, H. BOUTTIER and BERTRAND, Ann. de méd. **10**:173 (Sept.) 1921.

The report relates to a case of progressive bulbospinal myasthenia of the Erb-Goldflam type. (Previously reported by Marie and Bouttier in the *Bull. et mém. Soc. méd. d. hôp. de Par.*, April 30, 1920.)

In the present anatomicopathologic report, two points are of great importance.

Systematic examination of the central nervous system did not yield evidence of a nervous lesion. In particular, examination of bulbar, protuberantial and peduncular nuclei demonstrated integrity of their constituent cells. Thus, one can observe the classical picture of an Erb-Goldflam progressive bulbospinal myasthenia in the absence of nuclear changes.

In contrast to this integrity of the nerve centers, there were lesions throughout the suprarenal and muscular systems. Small lymphoid nodules were disseminated through the muscles. The suprarenal lesions were in the medullary zone and reticular substance.

The authors conclude that Erb's myasthenia rests on suprarenal-muscular system disease, the precise nature of which they do not try to give.

DAVIS, New York.

PALLIATIVE CONTROL OF THE GASTRIC CRISES OF TABETIC NEUROSYPHILIS BY THE RECTAL ADMINISTRATION OF CHLORAL HYDRATE AND SODIUM BROMID. ALBERT R. MCFARLAND, J. A. M. A. **78**:786 (March 18) 1922.

In the effort to devise an improvement on the morphin hypodermic for the relief of the patient with gastric crisis the author tried many drugs, using different methods of administration. He found that large doses of chloral hydrate and sodium bromid by rectum gave the best results.

The injection is preferably given at bedtime and consists of an aqueous solution of chloral hydrate and sodium bromid so prepared that one-half ounce (15 c.c.) of the fluid contains 40 grains (2.6 gm.) of each drug. The patient's hips are elevated by a pillow, and the solution is allowed to flow slowly under gravity pressure through a catheter introduced 15 cm. (6 inches) into the rectum. The patient is instructed to retain the injection. The room is then darkened, and the patient is kept quiet in bed. For persons weighing over 150 pounds (68 kg.), as high as 60 grains (3.9 gm.) of each drug at one dose can be given.

Of the twelve patients who were treated by this method nearly all had obstinate and severe cases. In all, forty-nine injections were given and definite relief was obtained in 76 per cent. of the forty-nine administrations. The last injection was apparently as effective as the first. Gastric lavage was used in two cases and seemed to be a valuable adjunct to treatment, especially if there was evidence of gastric distention.

NIXON, Minneapolis.

Society Transactions

NEW YORK NEUROLOGICAL SOCIETY

Stated Meeting, Feb. 7, 1922

FOSTER KENNEDY, M.D., *President, in the Chair*

DYSSYNERGIA CEREBELLARIS MYOCLONICA, PRIMARY ATROPHY OF THE DENTATE SYSTEM. DR. J. RAMSAY HUNT.

Some years ago, under the title dyssynergia cerebellaris progressiva or chronic progressive cerebellar tremor, Dr. Hunt directed attention to a peculiar disorder of motility, which he regarded as a definite clinical type of nervous disease. This condition was characterized by generalized intention tremors, which began as a local manifestation and gradually extended to other parts of the voluntary muscular system. The extremities, especially the arms, showed the greatest degree of involvement. When this tremor disturbance was subjected to careful analysis there was found associated with it a disorder of muscle tone and of the ability to measure, direct and associate muscular movements, the clinical manifestations of which were dyssynergia, dysmetria, adiadokokinesis, hypotonia and asthenia. All of these symptoms, including the volitional tremor, which was an extreme expression of the underlying disturbance of muscle tone and synergy, indicated a fundamental disorder of cerebellar function. Dr. Hunt, therefore, regarded the condition, with its progressive tendency, chronic course and well-defined cerebellar symptomatology as "an organic disease caused by degeneration of certain special structures of the cerebellar mechanism, which were concerned in the regulation of the tonus and synergies of muscles."

Since Dr. Hunt's original publication, he has observed another group of cases which combined the symptomatology of dyssynergia cerebellaris progressiva and myoclonus-epilepsy—the progressive dyssynergia characteristic of a cerebellar disorder in association with epilepsy and myoclonus. As in the group of cases uncomplicated by myoclonus-epilepsy, the movements of the extremities showed the greatest degree of disturbance.

This form of dyssynergia, which Dr. Hunt would term dyssynergia cerebellaris myoclonica, he has also observed in association with Friedreich's ataxia, a combined cerebellospinal involvement, which is not infrequent in the history of cerebellar system disease. Careful investigations were made of the pathologic condition in one of these cases, which throw considerable light on the origin and anatomic basis of the cerebellar portion of the symptomatology. There was a primary atrophy of the efferent dentate system of the cerebellum, and this system was regarded as the essential neural mechanism underlying the production of cerebellar or intention tremor.

Clinical Summary of Four Cases.—CASE 1.—Myoclonus-epilepsy began at the age of 17, and was followed five years later by symptoms of dyssynergia cerebellaris progressiva—generalized intention tremors and scanning speech, associated with dysmetria, dysdiadokokinesis, hypotonia and asthenia. With the exception of epilepsy, myoclonus and dyssynergia, there was no evidence of organic disease of the nervous system. The dyssynergia was appendicular rather than trunkal in distribution, and the higher types of movement were

chiefly affected. There was no familial history of either myoclonus-epilepsy or cerebellar disease.

Case 2.—A girl, aged 19, developed myoclonus-epilepsy at the age of 7, associated with a progressive dyssynergia of cerebellar origin, affecting especially speech and the extremities. The chief disorder of the cerebellar function was of articulation and the movements of the extremities. The gait and general equilibrium also showed some disorder of cerebellar function which was accentuated by a severe type of myoclonia. The dyssynergia, while preponderantly appendicular in distribution, also involved the trunkal musculature to some extent. There was no history of the familial occurrence of either myoclonus-epilepsy or cerebellar disease.

Case 3.—In this patient the myoclonus-epilepsy, of twenty-six years' duration, was associated with symptoms of dyssynergia cerebellaris progressiva: cerebellar dysarthria, intention tremor, dysmetria and adiadokokinesis, affecting chiefly the volitional movements of the extremities. With the exception of myoclonus-epilepsy and dyssynergia, there were no symptoms of organic disease of the central nervous system. Symptoms indicative of multiple sclerosis, Friedreich's ataxia or gross cerebellar disease were not present. There was no familial history of either disorder.

Case 4.—A girl, 15 years of age, with nocturnal epilepsy, had myoclonia and slight cerebellar dyssynergia of three years' duration. This case represents a somewhat earlier stage of dyssynergia cerebellaris myoclonica. There is no family history of myoclonus-epilepsy or of cerebellar disorder.

Remarks.—The association of cerebellar dyssynergia and myoclonus-epilepsy in this group of cases suggests the occurrence of two independent nervous disorders in one person. Combined forms of the various congenital, familial and hereditary types of nervous disease are not uncommon, and these associations are especially frequent in the spinal and cerebellar groups of systemic diseases. Jendrassik, who has devoted particular attention to this subject, records many such combinations. Such combinations, however, are rare and show merely a predisposition to the two disorders in the same person and would not necessarily indicate an essential relationship between them. On the other hand, little is known of the pathology and localization of myoclonus. Its occurrence, therefore, in conjunction with a special type of cerebellar disease is not without interest and may have a deeper pathologic significance than is apparent.

Dyssynergia Cerebellaris Myoclonica Associated with Friedreich's Ataxia.—Dr. Hunt presents another group of cases in which myoclonus-epilepsy and dyssynergia cerebellaris progressiva were associated with symptoms of Friedreich's ataxia.

Case 5.—A man, aged 36, had had symptoms of myoclonus-epilepsy since his twenty-first year. Previous to this there were symptoms of Friedreich's ataxia and progressive cerebellar tremor which steadily progressed. Study of the pathology of the condition showed the typical spinal lesions of Friedreich's ataxia associated with primary atrophy of the efferent dentate system of the cerebellum, considerable diminution in number and extensive atrophy of the cells of the dentate nucleus, with secondary atrophic changes in the superior cerebellar peduncles.

This case differs from those described in the previous series in two respects: the association with Friedreich's ataxia and the familial incidence; a twin brother suffering from the same disease. The first symptoms of the cerebello-

spinal disorder made their appearance during the early juvenile period. The myoclonus-epilepsy did not become manifest until the patient's twenty-first year. The presence of the spinal symptoms of Friedreich's ataxia would mask many of the typical symptoms of cerebellar disease. Marked intention tremors and scanning speech indicated clearly, however, cerebellar involvement (cerebellar tremor), and this was confirmed by histologic examination.

CASE 6.—A man, aged 29 years, the twin brother of the patient in Case 5, had had symptoms of dyssynergia cerebellaris and Friedreich's ataxia for the last ten years, gradually increasing in severity. Myoclonia developed five years ago, and the epilepsy was only of three years' duration. This case, like that of his brother, presents symptoms of dyssynergia cerebellaris progressiva, myoclonus-epilepsy and Friedreich's ataxia in combination.

Histologic Examination of the Central Nervous System.—Summary of Pathologic Changes: Histologic study of the central nervous system of the patient in Case 5 showed the typical spinal cord changes in Friedreich's ataxia. There was atrophy of the posterior columns of the cord which could be traced to the nuclei of Goll and Burdach in the medulla. Atrophic changes were also present in the direct cerebellar tract of Flechsig and the tract of Gowers (ventral and dorsal spinocerebellar tracts). The columns of Clarke were atrophic, and there were also some changes in the area occupied by the spinothalamic tracts.

There was no definite atrophy of either the direct or crossed pyramidal tracts, and the slight pallor in certain areas could be ascribed to degeneration of other nonpyramidal fibers in this region.

Histologic study of the brain-stem and cerebellum revealed an extension into the medulla of the system atrophies of the cord—the direct spinocerebellar tracts. The atrophy of the posterior columns apparently ceased at their termination in the nuclei of Goll and Burdach for the fibrae internae arcuatae were well preserved and the corpus restiform well developed.

The other important and essential lesion was an atrophy of the motor cells of the corpus dentatum of the cerebellum and of their efferent neurons in the superior cerebellar peduncles. There was no atrophy of the other cerebellar systems and none of the nucleus ruber. The lesion was therefore confined to this short and important internuncial common path which conveys the motor impulses of cerebellar origin to the spinal pathways. The lesions were therefore both spinal and cerebellar. The spinal lesion was that common to Friedreich's ataxia; Dr. Hunt would regard the cerebellar lesion as a primary atrophy of the efferent dentate system of the cerebellum.

Primary Atrophy of the Efferent Dentate System and Its Relation to Dyssynergia Cerebellaris Progressiva (Progressive Cerebellar Tremor).—We may recognize these pathologic types of cerebellar atrophy: the cerebellar cortical type of André Thomas; the olivocerebellar type of Holmes and the olivorubro-cerebellar type of LeJonne and L'Hermitte. Dr. Hunt would add the primary atrophy of the dentate system. This is a systemic disease characterized by atrophy of the cells of the dentate nucleus and thinning of the superior cerebellar peduncles. All other important structures of the cerebellum—the cortex, the olivocerebellar and pontocerebellar systems—are intact. In medical literature there are a number of isolated facts tending to show the close connections between what the author terms the dentate system and cerebellar or intention tremor. The relation of intention tremor to the cerebello-

rubral system was emphasized some years ago by Gordon Holmes. Sanders and Touch have also reported cases of intention tremor in association with lesions of the dentate nucleus. In the experimental field, the work of Ferrier and Turner on monkeys has also shown the relation of the cerebellar or intention tremor to the efferent cerebellar system.

As the pathologic changes in the case just recorded are limited to the neodentate system of the cerebellar mechanism, it is only natural that the more recently acquired, and more highly differentiated, motor activities should show the greatest degree of involvement. This system is related to the neocerebellum and its recently acquired function. Dr. Hunt believes that for this reason the condition is predominantly appendicular in its manifestations and intention tremor so conspicuous a symptom. The neodentate portion of the efferent cerebellar system shows greater vulnerability to abiotrophic degeneration.

The clinical syndrome which we term myoclonus-epilepsy probably includes a variety of clinical and pathologic types. Lafora and Glueck, and more recently Westphal and Sioli, have described a chronic progressive disorder of the central nervous system, characterized by epilepsy, myoclonus and progressive dementia. In one case, typical cerebellar symptoms were present with scanning speech, cerebellar ataxia and tremor. Necropsy revealed in both cases a peculiar amyloid degeneration of the ganglion cells which was widely distributed in the various aggregations of gray matter in the brain and spinal cord.

An interesting case of cerebellar atrophy associated with myoclonus has been described by Haenel and Bielschowsky. In addition to the typical clinical picture of a cerebellar disorder, there were symptoms of paramyoclonus multiplex. Careful histologic examination revealed only an extensive atrophy of the cerebellar cortex, of the inferior olive and of the olivocerebellar tracts. Pathologically the atrophy was primary and confined to certain definite systems of the cerebellum and belonged to the olivocerebellar type of cerebellar atrophy. It is interesting to note that the essential lesions in this case were confined to the cerebellar cortex and the cerebellofugal system of the first order. The slight changes in the nucleus dentatus and superior cerebellar peduncles, Bielschowsky regarded as secondary in character, following atrophy of the cortical neurons. If myoclonus, therefore, has any functional relationship to the cerebellum, it may exist in association with both types of primary cerebellar atrophy—the olivocerebellar type described by Holmes and the atrophy of the efferent dentate system.

DISCUSSION

Dr. S. P. Goodhart: Dr. Hunt has added considerably to a rather rare set of cases. I have not had the opportunity to see so many. I think it would be interesting to consider Dr. Hunt's proposition, which he has sustained by histologic examination and theoretical hypothesis. He has taken an unusual conception of cerebellar involvement. Our own idea does not embrace myoclonic movements. It is suggestive that the cerebellum is associated with that type of movement as explained by Dr. Hunt. I think, however, it is well to consider the relationship of the corpus striatum in cases of progressive cerebellar dyssynergia. The corpus striatum is certainly involved in some cases, and it seems to me that before we can definitely place this syndrome there should be more investigation and checking of necropsy findings, giving special attention to the larger cerebellar system and taking the corpus striatum particu-

larly into consideration. In one case particularly, a definite lesion was found in the striatal wall, and there was also involvement of the nucleus dentatus. The peculiarities of the striatal movements, however, would make it unsafe to place the entire syndrome in the literature without considerable confirmatory evidence. I think that Dr. Hunt has given us an admirable conception and has put us in contact with a syndrome not often seen. I should like to know why Dr. Hunt associates this myoclonia, so frequently seen with epilepsy, with the cerebellar mechanism.

Dr. E. D. Friedman: Dr. Hunt found degeneration of the superior cerebellar peduncles in one case. If that is so, may I ask Dr. Hunt whether he has noticed any choreiform movements in his cases? Both Forster and Bonhoeffer have associated choreiform movements with lesions involving the brachium conjunctivum. No one to my knowledge has thought of associating lesions in this area with myoclonic movements.

Dr. J. Ramsay Hunt, closing: My patients did not have motor disturbances of the choreiform type. Myoclonus and intention tremor were present, but no chorea in the technical sense.

Replying to Dr. Goodhart, these cases were cited as representing a clinical and not a definite pathologic type. For example, we recognize myoclonus as having a wide range of symptomatology, and we also recognize epilepsy as having the same wide range of incidence. When these two manifestations appear in conjunction we recognize myoclonus-epilepsy. We do not know the reason for this association, as it is probable that several pathologic conditions are involved in myoclonus-epilepsy. Nevertheless, it constitutes a clinical type or syndrome. In this sense I would regard dyssynergia cerebellaris myoclonica.

Here is a group of patients of which six have come under my personal observation, who had myoclonus-epilepsy and a progressive cerebellar dyssynergia. The relation of these symptoms to each other cannot be determined at this time. My pathologic studies of one case, associated with Friedreich's ataxia, showed a primary system disease of the cerebellar mechanism: atrophy of the dentate nucleus and of the superior peduncles. The cerebellar symptoms I would refer to atrophy of the dentate system. This is the chief efferent system of the whole cerebellar mechanism, and bears the same relation to the cerebellum that the pallidal system bears to the corpus striatum. The relation of myoclonus to a cerebellar disorder is not without interest. In Bielschowsky's case there was a pure system disease which involved the cerebellar cortex and the connections with the dentate system. There were also atrophic changes in the dentate system, but these Bielschowsky regarded as secondary. It is, however, significant that my case which involved the dentate system with its motor neurons of the second order, and Bielschowsky's case which involved the motor system of the first order, were both associated with myoclonus. So far as I am aware, this symptom has not been reported with other types of cerebellar system disease.

I refer to my conception of the static and kinetic system of motility, and to the cerebellum as the essential organ for the control of static or postural synergies. It is conceivable that disorders of the posturing mechanism might give rise to myoclonic types of movement, the result of a sudden break in postural control, the myoclonus being a compensatory reaction to sudden loss of postural control. This is purely speculative but is worthy of consideration in attempting to interpret myoclonus in association with cerebellar disease.

SOME RECENT EXPERIMENTS ON THE NATURE OF THE NERVE IMPULSE.

Dr. E. Newton Harvey, Princeton University (by invitation).

Although it is quite certain that nothing flows along a nerve, something is transmitted—the nerve impulse. Galvani (1786) and du Bois Reymond (1843) identified the impulse with electricity, but Helmholtz' (1852) measurement of the velocity indicated a process too slow to be compared with the flow of electricity in a wire.

Is the process physical (comparable to a wave motion like sound) or chemical (comparable to an explosion trail in gunpowder)? In general, physical processes are little affected by temperature; chemical processes increase two or three times for a 10 degree rise in temperature. Wave motions suffer diminution as they progress; explosion trails are self-propagative processes.

Experiments indicate that within normal limits the rate of the nerve impulse is greatly affected by a rise in temperature. This can be most easily studied in a jellyfish, Cassiopea, made of a disk of tissue covered with muscle and a nerve network. Rings of tissue like a doughnut can be cut from the disk and a nerve impulse started in one direction which passes round and round the disk with a velocity of about 0.5 millimeter a second. The impulse is entrapped and can be seen moving around because the muscles are stimulated to contract with each revolution. For the 18 to 28 degrees of temperature interval, the temperature coefficient Q_{10}, is 2.4.

Such rings inform us whether the impulse is self-propagative and will move forever. I have kept a nerve impulse moving in such a ring for eleven days continuously with no change in rate. The impulse traveled 537 miles. It stopped after eleven days only because regenerating tissue gave rise to impulses which counteracted the entrapped impulses.

The evidence indicates that the nerve impulse is at base a chemical process, although no chemical changes can be directly demonstrated. The action current shows that electrical changes are important, so that we may regard the impulse as electrochemical in nature.

Lillie (1918) has pointed out a perfect analogy in the transmission of a wave of activation in passive iron. Passive iron is iron so treated that it fails to dissolve in dilute nitric acid. It behaves like a noble metal. If touched with ordinary or active iron, it becomes active also, and the activity is transmitted from the point touched as a wave which can be followed by the eye. The following characteristics are common to both nerve transmission and propagation of activity in passive metal wires: 1. The wave is a propagation of negativity or negative potential, and the negativity at one region activates ("stimulates") the conductor at the next region. 2. All kinds of stimuli (mechanical, chemical, electrical) will activate. 3. Activation by the electric current is a polar phenomenon occurring at the cathode (Pflueger's law). 4. Activation by the electric current is more effective as the current is suddenly increased in intensity (du Bois Reymond's law). 5. The phenomenon of electrotonus appears in passive iron as in nerve. 6. After activation, a refractory period exists when another impulse will not be transmitted. 7. Inhibition phenomena appear in passive iron as in nerves. 8. Unidirectional passage of a synapse is observed with passive iron and nickel. The wave of activation is easily transmitted from passive Ni (sensory) to passive Fe (motor) but not transmitted from Fe (motor) to Ni (sensory).

DISCUSSION

Dr. D. J. Edwards, Cornell Medical School (by invitation): It has been interesting to listen to Dr. Harvey's direct presentation of the puzzling problem of neurophysiology. It has been customary for physiologists to lay out in a painstaking way the elements of conduction and the conditions that modify conduction, but they have always passed quickly over the matter of the propagated disturbance in the nerve. Dr. Harvey has attempted to solve that problem. One feature of the discussion has been the application to a fundamental physiologic problem of research in a field which appears at first to be entirely unrelated. Attempts at elucidation of the problem show how difficult it is to predict the ultimate application of fundamental research. Another feature of this discussion has been the relatively rapid development of this subject in recent years. About seventy years ago Johannes Mueller asserted that we should never be able to measure the passage of the nervous impulse. Five years later Helmholtz demonstrated it by a method so simple that every first year medical student may prove it. Some years later Fillié called attention to the minimal oxygen content necessary for propagation of the nervous impulse, and Waller called attention to the carbon dioxid output of the nerve when it is undergoing conduction. These observations laid a background for the chemical conception of the nervous process. A few years later Keith Lucas began his researches and laid the foundation for the present work, by showing that the propagation is without decrement, that is, the process goes on without being reduced; this has led to the chemical conception of the nervous process which Dr. Harvey has emphasized and brought out. The importance of the bearing of certain physical analogies on the nature of the process has been applied with great exactitude in the result of Dr. Harvey's researches.

Dr. Joshua Rosett: I am gratified to find that the latest work in physiology has resulted in a conception of the nature of the nerve impulse that is very much like the conception formed by Herbert Spencer and which always appealed to me as sound. Spencer discussed the subject over forty years ago, and his line of reasoning is almost the same as that followed by Dr. Harvey in his presentation. Spencer, too, rejects the hypothesis of an electrical nature of the nerve impulse as untenable, mainly on the grounds of the nondissipation of the nerve impulse as it travels along the fiber and because of the vast difference in the respective rates of propagation of the two impulses. The excellent illustration employed by the speaker of a wave of isomeric change in a passive iron wire, brought about by a small disturbance, is paralleled by an illustration employed by Spencer in connection with the same subject. Spencer speaks of the transformation from black amorphous to white crystalline antimony in a wire of that metal occurring when a slight disturbance—a light tap, the application of a hot object, etc.—is communicated to the wire at any point.

Dr. Harvey (closing): Herbert Spencer's idea is a very similar one, and it differs apparently only in this—in the case of antimony the whole wire changes from one allotropic form to another, but in the passive iron phenomenon the iron becomes coated with some sort of oxid film which makes it take a different potential to ordinary iron when the two are connected. If we believe that white antimony has a different potential to black antimony, we shall have an electrical change and therefore a similar analogy.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Monthly Meeting, Feb. 16, 1922

F. H. PACKARD, M.D., *President, in the Chair*

AN EXPERIMENTAL STUDY OF THE MECHANISM OF HALLUCINATIONS. MORTON PRINCE, M.D.

Dr. Prince presented the results of an experimental research in the mechanism of hallucinations. All the traditional theories, he said, which could be classified in two groups—anatomicophysiologic and the psychologic—were unsatisfactory. As the result of previous study Dr. Prince had reached the conclusion that visual hallucinations were the emergence into consciousness of normal imagery pertaining to subconscious processes. The method of investigation previously employed by him was that of introspection in hypnosis, by which memories of subconscious processes were obtained. It remained to prove this theory by objective methods.

Such a research required a subject who exhibited both visual hallucinations and synchronously occurring subconscious processes which could be "tapped" by methods permitting physical records of the same to be obtained, namely, so-called automatic, that is subconscious, writing. Such a script, of which the subject would not consciously be aware during its production, would obviously be a physical record of ideas occurring subconsciously, and if an hallucination occurred synchronously its imagery might be correlated with the ideas of the script if the theory was valid.

The technic of the experiments was as follows: The head of the subject was covered with an opaque cloth to prevent her seeing the script as it was being written automatically by her hand. A pencil was then put into her hand which rested conveniently on a sheet of paper placed on a writing tablet by her side. She was then told to write automatically regarding some subject which was designated in general terms in each experiment: for instance, a memory of some remembered episode in her life; a memory of such an episode, but one forgotten by the subject; a fantasy; a fabrication requiring constructive imagination. The object of diversifying the subjects was to obtain products of different kinds of subconscious work (memory, dream-like fantasy, imaginations, etc.). If, during the experiment while the hand was writing an hallucination developed, the subject was directed to indicate the fact the moment she saw it by exclaiming, "picture." Thereupon a mark was made on the script at the point where the picture appeared. Likewise the moment the hallucination disappeared the subject exclaimed as directed, "gone," and the point was similarly marked on the script. Thus those words of the script which were written during the occurrence of any given hallucination could be identified and could be compared with the latter and any correlation of the written ideas and the hallucinatory images noted.

Under these conditions it was found that the writing of the script by a subconscious process was accompanied by a synchronously occurring hallucination.

After the observation was complete the script and the hallucination as recorded were compared and for this purpose arranged in parallel columns. Thus any correlations between the imagery of an hallucination and the synchronously written script could easily be noted. (The paralleled results, script and hallucination, were exhibited on the screen by lantern slides.)

On comparing in this way the description of the hallucination with the script it was easy to recognize that the images of the hallucination corresponded with the ideas recorded in the script and were such as normally would be the imagery contained in those ideas. For example: The script described a memory of a particular scene in a room at "Harvard University," and the imagery of the visual hallucination was an exact reproduction. The script was a verse describing in allegory a treasure chest, a fountain, golden musical instruments, etc., and the synchronously occurring hallucination was a perfect visual representation of those ideas, such as normally would be the case. The script described a fabricated romance and the personages of the romance and the local scenes were faithfully represented by the visual imagery of the hallucination; and so on.

After the observations were made the subconscious process was directed to answer in writing a questionnaire directed to ascertain what kind of process occurred subconsciously during the hallucinations and the relation of the one to the other. The script testified that during the production of the script images corresponding to the written ideas appeared first subconsciously, without the subject being consciously aware of them, and that then those images emerged into awareness as the hallucination.

A second series of experiments was made, the converse of the preceding; that is to say, hallucinations were primarily produced by fixation of the attention and the automatic script employed to determine what kind of subconscious process, if any, and its content, occurred synchronously. The results were the same as in the preceding series.

Auditory hallucinations were found to have the same mechanism, as was also the case with the imagery of a dream.

The following conclusions were drawn:

1. There is a type of visual hallucination in which the imagery has its source in a dissociated mental process of which the subject is not consciously aware. Such a process is by definition a subconscious one.

2. The content of this subconscious process contains images identical with the normal imagery of conscious thought.

3. The hallucination is due to the emergence into consciousness of the previously subconscious images. This emergence necessarily results in a hallucination in that the imagery of the latter is not related to the content of the conscious train of thought, but is foreign to the latter. This is a necessary consequence of the imagery being normal elements in a separate dissociated train (mental process).

4. The subconscious process is essentially a conscious one of thought.

5. There is a type of auditory hallucination which has essentially the same mechanism.

6. As there is a type of hallucination (visual and auditory) occurring in the insanities which is identical in form, structure and behavior with that produced experimentally in this study, the conclusion is justified that such hallucinations of the insane are due to the same mechanism.

7. The implication follows that when hallucinations of this type occur in the pathologic psychoses, they are indications of the activity of a dissociated subconscious process as a factor in the psychosis.

8. The hallucinatory phenomenon carries the further implication that the genesis and psychopathology of the psychosis are to be found in the forces which have determined the dissociation and motivated the subconscious process.

9. It is not to be assumed that all hallucinations have the mechanism of the type here studied. It is possible that in those occurring in the intoxication psychoses and in certain forms of organic brain disease, particularly when the hallucination is of a simple unelaborated static structure, the imagery is induced by direct irritation of the cortical or subcortical neurons. It is difficult, however, to exclude the possibility that the intoxicating agent or organic process simply removes inhibition and permits subconscious dissociated processes to function. Nor can we find any analogy with the known effect of irritation of motor and other areas of the brain. Irritation, as observed, produces simple movements and simple sensory phenomena (noises). Still, the possibility of irritating factors becoming the immediate excitants of organized complexes of neurons underlying the hallucinations, cannot be excluded. This theory, however, needs to be proved. Even the irritative theory, as opposed to the psychogenetic theory, permits the interpretation that the irritation excites a dissociated subconscious process from which images emerge into consciousness.

10. The psychologic problem of differentiating between normal imagery and hallucination disappears in that they are identical, the hallucination being only the normal imagery of a dissociated subconscious process.

11. If the evidence given by subconscious introspection be not accepted, a possible interpretation of the hallucinatory imagery is that the images do not themselves occur primarily as subconscious elements, but by the same mechanism appear in awareness as the conscious correlates of a coactive dissociated physiologic process; in other words, a subconscious process is neural, not psychic. On the other hand, such an interpretation does not take into account a large mass of collateral evidence for the psychical nature of processes occurring outside the field of awareness.

12. So far from a hallucination being a regression to an infantile form of thought (Freud), it is an element in highly developed adult thought processes.

13. The mechanism of the imagery of some dreams is the same as that of the hallucinations of the type here studied.

DISCUSSION

DR. DONALD GREGG: How can you determine that what is being written is unknown to the person?

DR. PRINCE: In one sense you cannot, any more than you can tell when a person comes into your office and says that he has a pain in his back that he is telling the truth and is not a malingerer. We must bear in mind that automatic writing, like lumbago, is a common phenomenon and has been observed and recorded by a large number of experimenters in all countries. It is, therefore, a well established phenomenon. It is always possible that a person may be a malingerer and the question, if raised, must be determined as in the malingering of other conditions. Let no one imagine, however, that medical men and scientists generally have a monopoly of integrity. The particular subject who was used in these experiments had written a large amount of material long before she came under my observation . She wrote automatically for her own pleasure. A number of investigators have trained themselves to write subconsciously and their testimony is unimpeachable. It is not difficult for some people to acquire this ability. In an audience like this, no doubt we could find two or three automatic writers, given proper conditions; then after say a month's practice and training we should probably find that a fair percentage of those present could produce automatic script.

DR. DONALD GREGG: In this particular case, did the subject know what she was writing?

DR. PRINCE: No. Automatic writers differ very much in this respect. Some have no awareness at all of what the hand is writing and indeed may not know that the hand is moving at all, for in some cases the hand becomes temporarily anesthetic during the production of the script, as was first shown by William James. In other cases the written ideas of the script emerge into consciousness during the writing and yet it is automatically or subconsciously produced, as is shown by the fact that if the writing is stopped at any given moment the writer is found to be ignorant of what is going to be written and cannot consciously finish the sentence and the theme.

Whether or not the subject is aware at the moment of what the hand is writing is not in principle of importance for the script is still produced automatically. It may be of importance only for the particular experiment that is being made. Then, again, writing may be obtained from a person who had previously had complete anesthesia of the hand. The subject therefore cannot tell through the muscular action what the hand has written or know that the hand has moved at all.

An important phenomenon, frequently observed during subconscious writing when the subject is unaware of the written ideas, is the emergence, not of the ideas, but of the affect pertaining to the subconscious process. The affect of exaltation or joy, or depression or fear, may thus emerge into consciousness when such an affect does not belong to the conscious thoughts of the writer but can be traced to the subconscious process. This phenomenon I have observed hundreds of times under experimental and other conditions. I believe this to be the mechanism of the emotional state in many of the psychoses, such as manic-depressive states, some phobias, and even in many normal conditions. The affective condition is due, as I have said, to emergence into consciousness of the affect belonging to a dissociated subconscious process.

Question by a Member: Can a subject perform another action at the same time that he is writing?

DR. PRINCE: In general, yes. But the performance of the action differs somewhat with its nature. He can perform all ordinary movements, subject of course to the necessity of writing at the same time. He can observe accurately everything going on about him, such as watch and recognize accurately and without confusion what is passing in the streets, comment on and enjoy the situation, read aloud from a book, and so on. In the last instance, however, he is liable to go into a dreamy state, in which he keeps on reading. The trouble here is, I think, that it is difficult, even if possible, for two different processes to make use of the language function at one and the same time. There is apt to be blocking of one by the other. On the other hand, according to my experience, an automatic writer can passively observe, think freely and reflect on what he observes while the automatic writing is being produced. During the writing by such subjects as I have observed, it was difficult for the subject to find the precise language in which to express his conscious thoughts. In other words, he seemed to be largely robbed of the language function by the subconscious writing process.

DR. D. A. THOM: Were the emotions of the subject appropriate to the hallucination she was going through?

DR. PRINCE: Yes: In this connection, as an example of the emergence of the mood belonging to the subconscious processes, I may cite one observation in this study which is of interest.

When the subconscious system wrote as a joke a pretended spirit message and while this system, under interrogation in regard to it, manifested through the script, a humorous, almost hilarious mood, the subject herself without obvious reason was consciously in high spirits, joyful, and felt the spirit of fun. Later, during the latter part of the examination, when the subconscious system wrote bitterly and remorsefully of the past, confessing subconscious sins, the subject remarked during the production of this script, without knowing what the hand had written, "I feel serious now. All my high spirits and feeling of fun have left me. I have a sad, remorseful feeling."

I am astonished that psychiatrists have not made use of induced hallucinations in order to study the mechanism of hallucinations in the insane. Here is material and the method right at hand for experimental studies. The observations can be controlled and the hallucinations traced to their origin, and we have at least an approach for an attack of this and many other psychiatric problems. Likewise automatic writing is a most valuable means of research.

Question by a Member: Is it as easy to get automatic writing from the insane as from normal people?

Dr. Prince: In reply to the question just put to me I would say, unquestionably no. It is necessary to have complete cooperation between the subject and the experimenter, and in most insane people that is impossible. Then, too, the dominating mental processes of the insane block the impulses of any subconscious processes that might be stimulated to write automatically. On the other hand, there are many borderline cases, like those of the early stages of dementia praecox, in which automatic writing might be easily obtained and the experiments of the kind I have mentioned carried out.

Dr. E. W. Taylor: How about febrile delirium? I remember with interest a study which you made some years ago of a postpneumonic delirium in which you were able to explain absolutely the character of the hallucinatory phenomena that resulted.

Dr. Prince: Yes. It was a case in which during the delirium there were in activity normal conscious processes which perfectly oriented the situation, recognized clearly the delirious character of the conscious process and fully explained the delirium, and hallucination. I have observed this phenomenon several times. In this connection I may mention an experiment which I made comparatively recently in the case of delirium during typhoid fever and continuing during convalescence. I was asked to relieve the insomnia present by hypnotic suggestion. As I had a suspicion as to what might be the psychogenetic cause of the delirium I took the occasion to make the experiment, although I had little expectation of success at the time. While the patient was in hypnosis I made various explanations and suggestions along the line of the theory which I had formed as to the real cause of the delirium. The patient was living at that time in the country. I left him fast asleep and returned to Boston. I had hardly reached my house before the telephone rang and to my surprise the patient was at the other end and reported, to his great delight, that he had entirely recovered, that he recognized the delirious character of his ideas and that he was all right again. His delirium disappeared for good, and to my surprise my experiment was a success. My explanation is that the delirium at the time of the experiment was entirely functional. Whether this was its nature during the fever it is, of course, impossible to say, but if not it must have become transformed into a functional delirium. In the case of pneumonia, to which Dr. Taylor referred, I think it must have been a functional delirium from the beginning.

DR. E. W. TAYLOR: Do you believe that such a method could be used generally?

DR. PRINCE: I think not, for the same reason that the method is not practical in most cases of insanity. Perhaps, however, it might be used more than we think.

DR. F. H. PACKARD: I take it that you meant the toxic condition had disappeared when the experiment was made, that there would be more chance of success under those circumstances than during the toxic period.

DR. PRINCE: Precisely, if the primary delirium is really due to the effect of the toxemia. But I think that this pathology may be fairly questioned. To my way of thinking it is more probable that such delirium is due to the loss of inhibitions. The toxemia paralyzes the normal inhibitions thus permitting the abnormal processes constituting the delirium to take place, somewhat after the fashion of dreams.

DR. E. W. TAYLOR: In the hallucinations of smell which we occasionally see in tumor of the temperosphenoidal lobe, there certainly appears to be a structural origin.

DR. PRINCE: I think a simple sensory phenomenon might be obtained from a structural origin, such as a smell, a flash of light, or a noise like buzzing or whizzing, but not a complicated process like a visual picture or the musical strains of grand opera; that is to say, elaborate ideas with meaning.

DR. E. W. TAYLOR: The original stimulus is certainly organic, whatever the elaboration of it may be.

DR. PRINCE: As I have said, you may and do, undoubtedly, get simple sensory and motor phenomena from the pure stimulation of an organic process, but do you ever get elaborate hallucinations? I doubt it. Nor have I been able to find in the literature any accounts of such cases. Organic irritation of a motor center will produce simple movements like the flexion or extension of a limb: but did you ever hear of an organic irritation or a motor area exciting the complicated movements of a baseball pitcher—spitting on the ball, rubbing his hands in the mud, winding himself up and then delivering a curved ball to a "Babe Ruth"? That is practically what we are asking an organic process to do in producing analogous sensory and ideational phenomena by this theory.

DR. HUGO MELLA: What is the theory of the mechanism of the suppression of these inhibitions in cases of the nontoxic variety?

DR. PRINCE: In such cases we have conflict, repression and consequent dissociation followed by the motivation of the dissociated system as a subconscious process by some strong anxiety, wish, aspiration or other impulse. The original breakup in this patient, for example, was due to a conflict between two sides of her nature, one longing for another, an intellectual life, believing that she had musical and other talents and hating the banal life she was obliged to follow; the other side motivated by the ordinary sense of duty and obligation and certain urges. It would take us too far away to go into these.

In reply to the question by a member whether I "asked the subject a question which was really directed to the subconscious and answered by the hand," I would say that this inquiry raises a complicated problem. As ordinarily expressed, I asked the hand, or the subconscious, a question. This, of course, while the subject was not hypnotized. That seems a paradox. It is,

of course, a figure of speech. And, after all, it is a figure of speech to speak of the subconscious as a separate mind. We are really dealing with different mental systems or complexes of which the mind is constructed and which are more or less closely integrated into one mind. They are all one mind, and there is only one mind, however much the different systems of which it is composed may be temporarily disintegrated. In the latter condition the systems are out of gear, out of harmonious integration and cooperation. And yet by simply talking to a person you can reach the dissociated systems. It is not necessary to hypnotize the subject and bring these systems into the content of conscious awareness. Let us speak in terms of stimulus and reaction. A given stimulus strikes and awakens a reaction from a system which is so organized and attuned to it that it will react. Otherwise no reaction will follow. Thus, for example, if the composite content of conscious interest is solely occupied with thoughts and affects of a baseball game, a stimulus suggestion related to religion and going to church will awaken no reaction from the baseball content of conscious awareness, but it will, or may, strike and awaken a reaction from a subconscious system of religious memories, ideas and emotions, even though it be dissociated and though the memories be consciously forgotten. In practical therapeutics this principle is of the greatest value. However it be explained, the fact remains that questions and suggestions can stimulate and awaken a reaction from a dissociated system even though they have no meaning for the content of conscious awareness of the moment. The personality can be compared, in a way, to a complex manufacturing establishment, like the Ford plant. There is the casting department, the department where cylinders, gearing, bolts, nuts, wheels, bodies, etc., are made, and the assembling department. All are under a central executive control. Accordingly they all work harmoniously. But sometimes a strike occurs in one of the departments; the cylinder department refuses to cooperate and make cylinders. Not only efficiency is impaired, but economic adjustment of production of the integral parts to the total output is disarranged. The executive must get in touch with the group of strikers. He can do so without disturbing the other departments. Any questions, directions, or appeals to the strikers will not awaken a response from the departments that are at work. In the same way we can have a strike in any of the different systems of the mind and it can be settled in the same way, by arbitration, conciliation, persuasion or suggestion.

Of course our knowledge of the integration of the systems of the mind is superficial, and any explanation is incomplete. We can hold, however, to the empirical facts of experience.

As to psychotherapeutics: It is an art, not a science, though it makes use of scientific principles. This is one of the difficulties in teaching it. You can teach principles but their application is an art that comes from experience, like painting a picture.

As to the principles, what I am now going to say may sound arrogant, but I am going to say it: To investigators who have had long training in psychologic phenomena and particularly subconscious phenomena, and are familiar with them from personal observation and experimentation, as a bacteriologist is trained in bacteriology and a physicist in physics, the Freudians for the most part seem like amateurs, because very few, if any, show by their writings that they are familiar by personal training and experience with all the phenomena of the subconscious. What would you think of a person who undertook to formulate laws regarding the electrons, atoms and electricity

and determine the ultimate nature of matter, who had only read the writings of physicists and whose own experiences were limited to the phenomena obtained from an amateur's wireless outfit, or to studying the discharge from a particle of radium or a roentgen-ray machine? Even Freud, himself, has never shown by his writings that he possessed the requisite training and experience, though he may not have disclosed it. Indeed he has repudiated the use of methods of research which others have found essential. One must know all the phenomena of the mind and not only those obtained by a single method of research like that of "free association" if one is to reach sound conclusions regarding the laws of the mind.

CHICAGO NEUROLOGICAL SOCIETY

Regular Meeting, Feb. 16, 1922

C. F. READ, M.D., *President, in the Chair*

AN UNUSUAL CASE OF PARALYSIS AGITANS. HUGH T. PATRICK.

A physician, 52 years of age, has suffered from unilateral paralysis agitans sine agitatione for approximately two years. Tremor occurs occasionally in the arm when the patient is tired, and is of intention type. The unusual feature is the unilateral involvement of the face. The lips on the unaffected side open more widely in talking (the exclamation point mouth of Charcot) and in smiling, and the right eye closes more than the left. Such affection of the face is not so uncommon as I at first thought, for I find records of four similar cases among the histories of patients seen in my office. In two of these the right and in two the left side was involved. I believe there is no real paralysis in these cases, but that the muscles are involved by the peculiar rigidity of the disease and consequently are less responsive.

In the first of the four patients referred to, seen in September, 1899, it was some time before I discovered paralysis agitans, and the method of discovery is of some interest. In testing the strength of the unimpaired hand and forearm I asked the patient to grasp my hand firmly and supinate and pronate his own hand against my resistance. When he did this, the other, supposedly paretic hand, assumed the position of, and began to shake with the tremor of, paralysis agitans. I have never seen this method described in the literature, but it sometimes brings out the tremor when nothing else will. This patient also distinctly had involvement of the face. In maintaining the forehead wrinkled the right (affected) side would tire; then it would let down and the forehead on that side became smoother. In showing the teeth the right side lagged a little, and when he wrinkled his nose to "make a snoot" a tremor started on the right side of the face.

SPECIMEN OF CYSTIC TUMOR OF THE LEFT FRONTAL LOBE. PETER BASSOE.

Three years ago following an injury to the upper jaw, a man, 26 years of age, had a discharging sinus in the gum. In July, 1921, he began to complain of frontal headache; in September, he had to be confined to bed; and in October, he became semistuporous. A few days after admittance to the Presbyterian Hospital he was shown in a clinic; he claimed that he was at the

stock show. Examination revealed bilateral choked disk, the swelling on the right being 2 diopters and on the left 3.5 diopters. A bilateral Babinski sign was present, but the reflexes were otherwise normal. There was no paralysis or sensory disturbance. A cyst in the upper jaw containing creamy pus was evacuated, following which the headache was less severe. Further relief was obtained after lumbar puncture, which yielded a clear fluid under increased pressure (25 mm. mercury). All tests of the fluid, including the Lange test, were negative. Visual form fields were normal, but there was a contracted color field in the right lower quadrant. On January 3, the swelling in the right disk was 3 diopters; in the left, 4 diopters. On January 5, Dr. C. B. Davis punctured both lateral ventricles through the occipital bone. No fluid was obtained on the left, but fluid spurted out from the right side under increased pressure. The patient was stuporous most of the time both before and after this operation. Weakness of the lower facial muscles on the right side became distinct. A tentative diagnosis of tumor or abscess of the left frontal lobe was made, and an operation planned for February 2. Coma developed during the preceding night and respiration ceased just before the time set for the operation; after two hours of artificial respiration the heart also stopped.

The brain showed in the left frontal lobe a cyst, 6 cm. in diameter, not connected with the ventricle but having a smooth lining which resembled ependyma both macroscopically and microscopically. At the other end of the cyst there was a solid gray tumor, 3 cm. in diameter, which reached the anterior and mesial surface of the brain where the dura was adherent. The tumor was apparently an endothelioma.

DISCUSSION

DR. LEWIS J. POLLOCK: I wish to call attention to the fact that this specimen is somewhat similar to that I showed at the last meeting. If the ventricles were studied to determine whether the aqueduct of Sylvius is patent, the cyst might be entered, with consequent failure to recover the indigo-carmin by spinal puncture.

DR. HUGH T. PATRICK: Is there any explanation for the formation of a cyst in connection with an endothelioma?

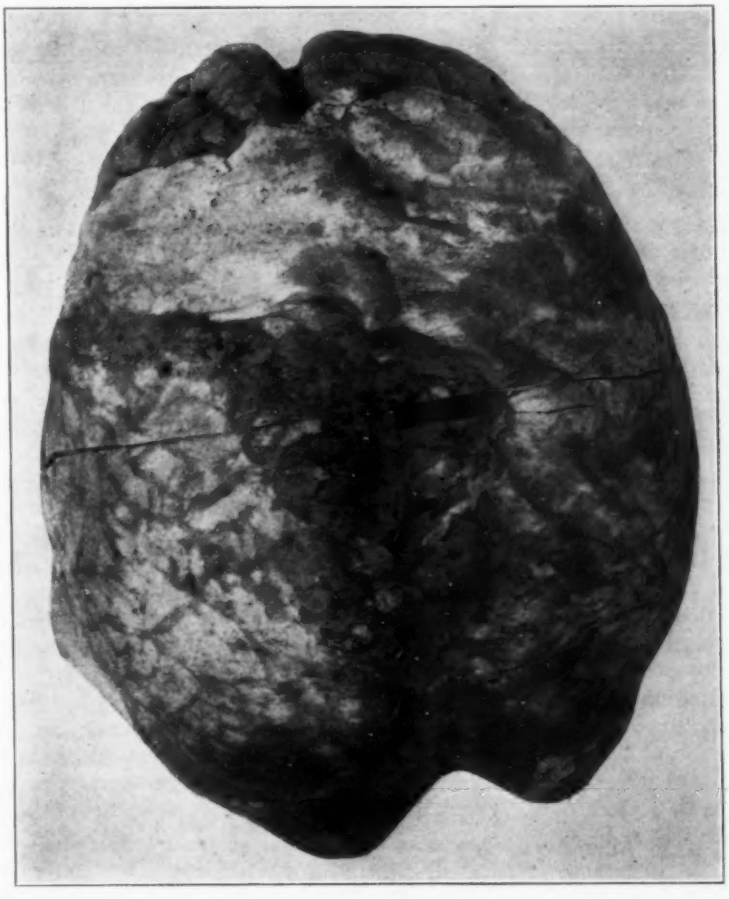
DR. D. B. PHEMISTER: An endothelioma associated with a large cyst is certainly uncommon. All brain tumors with accompanying cysts that I had seen previous to this were gliomas.

TUMOR-LIKE INFILTRATION OF CEREBRAL DURA: A CASE OF LYMPHOSARCOMA WITH CLINICAL PICTURE OF HODGKIN'S DISEASE. Presented by PETER BASSOE.

This brain was taken from a man aged 35 years at death, who had developed glandular enlargement of the neck four years previously. He was under the care of Dr. Billings in the Presbyterian Hospital much of the time, and he died there. A careful record was kept and much laboratory work was done in the case, which was considered one of Hodgkin's disease. Cultures and vaccines were made and glands removed for histologic examination. At times the enlargement of both lymph glands and spleen seemed to have disappeared, but always to recur. All groups of glands were successively affected. When last admitted to the hospital, five weeks before death, mental dulness and failure of memory were noted. There was severe pain in the legs ascribed to local lymphoid infiltrations. Three weeks before death there were twitchings of the arms and legs and increased mental disturbance, but there were no

convulsions or paralysis. A week before death an abscess developed in the left ear. Hiccup was frequent during the last two weeks, and vomiting during the last three days.

At necropsy, in addition to enlargement of spleen and lymph glands, there were erosion and lymphomatous infiltration of the sternum and of the skullcap. The dura was adherent, thickened, infiltrated with lymphoid tissue, and the site of numerous hemorrhagic spots having an appearance similar to that of chronic



Dural infiltration resembling chronic pachymeningitis in a case of generalized lymphosarcoma.

productive pachymeningitis. This "heterotopic" lymphoid infiltration, together with the general histologic features, indicates that the case should be considered one of lymphosarcoma rather than Hodgkin's disease.

DISCUSSION

DR. D. B. PHEMISTER: I think the lesion in this case was a primary involvement of the skull with secondary invasion of the meninges. The marrow of several other bones also contained lymphosarcomatous tissue which had broken

through the cortex. Against this view is the fact that the skull erosion was nearly all of the tabula interna. That would speak for primary meningeal involvement with secondary invasion of the bone.

AN ANALYSIS OF 146 CASES OF PARALYSIS AGITANS. HUGH T. PATRICK AND DAVID M. LEVY (by invitation).

This paper appears as an original article on page 711 of this issue.

DISCUSSION

DR. MEYER SOLOMON: With respect to the correlation between the location of the injury and the site of the first symptoms, one wonders whether the injury was not the result rather than the cause of the disease, the patient having difficulty in using that part of the body.

DR. W. G. STEARNS: In the last six cases I have had under observation a Wassermann test of the spinal fluid gave from + to +++ reaction in three.

DR. G. W. HALL: How did Dr. Levy arrive at the age? Is this the age at the time of consultation or of onset? Incidentally, it would seem that within a few years the age curve will have to be modified if the number of cases of so-called paralysis agitans following lethargic encephalitis is maintained.

DR. PETER BASSOE: From now on it will be of interest to study the histories of infections these patients have suffered from prior to the onset because of the occurrence of paralysis agitans after epidemic encephalitis. While at first the tremor is different, as time goes on the resemblance to true paralysis agitans becomes greater. Some patients apparently recover, and then paralysis agitans in typical form develops long afterward. I have seen two cases this year in which the symptoms began one and two years after the acute illness. One patient, a woman, had epidemic encephalitis in 1919 and in 1921 developed typical parkinsonian tremor in one leg. She is only about 25 years of age, and I think it is fair to say that the preceding encephalitis must be considered the cause. Recently Marinesco has expressed the opinion that in such cases there is an actual infectious process which becomes chronic, and thus the parkinsonian syndrome is not merely a sequel but is an active and progressive disease. Marie and other French observers find that a history of trauma is less frequent than a history of some acute disease. That is the weakness in Dr. Levy's statistics. As he said, they were made from records of what was considered important at the time the history was taken, and I assume those records did not give an account of all previous infections.

DR. A. B. YUDELSON: The symptoms in the postencephalitic cases differ essentially from those in paralysis agitans in that the rigidity is not as great in proportion to the tremor. In six such cases seen during the past year all patients had marked generalized rhythmic tremor. Although this was of the parkinsonian type, it was more generalized and not of the typical "pill-rolling" character. A history of a profound infection of prolonged duration is usually easily elicited and is very characteristic.

DR. DAVID M. LEVY, closing: The question concerning the site of the trauma can be quickly answered by going over a few of the case records. Age was taken at the time of onset and not at the time of examination. One man, aged 70, gave a history of falling and spraining the left arm. Two months later the disease started in that arm. A bricklayer, aged 48, fell from a building on his right shoulder. The right arm was limp and weak for six weeks; five years

later the first symptoms of paralysis agitans appeared in the right hand. A man, aged 56, hurt his right wrist in a boxing match. Roentgen-ray examination showed a dislocation of the scaphoid bone. The right wrist was first involved in the disease. So in all cases, the traumatized side, where trauma had occurred, was involved first.

A history of syphilis was given in only three cases. Past illnesses were not recorded with a view to finding a possible cause for the disease but in the usual routine manner. The commonest form of previous infection noted was rheumatoid arthritis. Of eight cases in which the first part involved was noted, in six this part was first affected by the paralysis agitans.

I have here dealt only with classical paralysis agitans and have excluded the postencephalitic types. I have no statistics on such cases, but I have grouped the cases of epidemic encephalitis by decades and in comparison with the actual population. Parkinson's disease as a sequel to this infection would be liable to occur in any group, and the curve should follow an irregular line like that of encephalitis.

THE MAKEUP OF THE NEUROTIC. By Meyer Solomon.

All of us are in a state of unstable equilibrium. The neurotic is in a state of relatively greater instability than the average person and must struggle for poise and equilibrium more constantly, vigorously and persistently. The problem reduces itself to a study of the causes and manifestations of the disequilibrium.

The disequilibrium of the neurotic is shown by these traits: (1) Relatively increased nervous instability—oversensitiveness and undue irritability, typically illustrated by excessive disturbance from sudden noises or excessive stimulation. (2) Over-suggestibility, which may be general or special. The former is evidenced by excessive credulity and uncertainty resulting in mental conflicts, distractibility and confusion. Special suggestibility leads to marked habit formations, stubbornness and domination by ideas. (3) Lack of self control. (4) Nervous and mental excitability, as shown by impulsiveness, emotionalism on slight provocation, etc. (5) Fatigue which results in functional symptoms—visceral, skeletal, cerebral, emotional or moral. (6) Nervous and mental tension with uneasiness, restlessness and exaggerated knee jerks. (7) Awareness of these conditions with resultant self-consciousness, doubts and obsessions. (8) Difficulties of adjustment in regard to physical health, nervous or mental condition, moral-ethical-religious relations, social contacts, family problems, politico-economic problems and special wishes. (9) Pathologic fears and convictions.

The neurotic struggles for poise and equilibrium by flight or fight. He employs various means, often bizarre, and often goes to extremes. Efforts at flight may lead to hysteria, exaggerations, timidity and fears. Fight on the other hand may cause depreciation of others, hate, jealousy, self-expansiveness, over-altruistic notions and psychoses, particularly paranoid trends. Drug addiction, medical fads and nonmedical cults, devotion to religious and philosophic interests, peculiar habits; excessive craving for sensation, excitement and change may also be adopted.

The neurotic person is more apt to have excessive and prolonged mental conflicts, which are then the result and not the cause of the neurotic condition. But mental conflicts may of themselves produce nervous upset or aggravate the neurotic makeup, a vicious circle resulting.

Individualization is essential in the study and treatment of neurotic persons.

PHILADELPHIA NEUROLOGICAL SOCIETY

Feb. 24, 1922

CHARLES H. FRAZIER, M.D., *President*

HYPERTROPHICA MUSCULORUM VERA. DR. TEMPLE FAY.

A man, 60 years of age, a full blooded negro, from Dr. Spiller's service at the University Hospital, had always been well and strong prior to the fifteenth year, when he was ill for five weeks with what was diagnosed as pneumonia, although typhoid fever could not be ruled out. Two years later his entire left side suddenly became paralyzed; the skin on this side was hyperesthetic. He gradually recovered, and at 19 was able to use all the muscles and to do the most difficult type of labor.

The patient was 47 when first seen at the dispensary in 1909. At that time he complained of cramp-like pains in the muscles, accompanied by gradually increasing weakness. His neck muscles were so weak that his chin rested on the sternum, and he walked with a marked compensatory lordosis. After walking half a mile he would begin to stagger, and his leg muscles became weak. He was unable to lift heavy weights and had feeble power in the arms and shoulders, though these muscles appeared large and powerful. The trapezius, deltoid, biceps and triceps muscles were enormously developed and when contracted felt firm and hard. He had been doing hard laboring work for twenty years, but six years previously he first noted loss of strength. This gradually progressed until he became very weak in all his muscle groups, and was unable to work.

As muscular weakness appeared he suffered with severe pains and cramps, chiefly in the extremities. At this time his reflexes were prompt and equal. The Babinski sign, ankle clonus and Romberg sign were absent. The irides reacted normally to light and accommodation. He complained of difficulty in swallowing, and there was slight weakness of the bladder sphincter.

A specimen of muscle was removed in 1909 and carefully studied. The fibers were round in cross section and ranged from 80 to 180 microns, the average being 130 microns. The normal diameter of fibers from similar muscles is from 60 to 80 microns. There was no excess of fat or connective tissue found, and the muscles showed true hypertrophic changes.

The muscles, tested with the faradic current for two minutes, showed no reactions of myasthenia. An interesting test was carried out at Dr. Wood's request by Dr. McKenzie, who gave the patient the usual gymnasium tests. He did not reach the average of students who were of his muscular development, but fell far below the average of students who lead the most sedentary life.

Dr. Andrew H. Woods presented him before this Society in 1910, reporting the case in the *Journal of Nervous and Mental Disease*, Vol. 38, No. 9, 1911, myasthenia gravis, Thomsen's disease and pseudohypertrophic muscular dystrophy having been suggested as possible diagnoses. Electrical findings ruled out myasthenia gravis as there was no decrease in reactions of the muscles to strong faradism. He had no difficulty in starting voluntary movements and immediate relaxation of the muscles after a movement was completed, thus showing none of the typical signs of myotonia congenita. The microscopic findings of the muscle tissue were not those found in the pseudohypertrophic form of muscular dystrophy.

The patient still shows marked involvement of most of the muscle groups, especially those of the back, arms and legs, without atrophy. The anterior neck muscles are not hypertrophied. The Achilles reflex is absent on both sides, but the patellar reflexes are prompt. The plantar reflexes, station and sensation are normal.

The case is interesting because the progress of the disease in the last twelve years has not been marked. The calf muscles show slightly more enlargement at the present time than they did in 1909. For a period there was improvement, and he was able to take care of a garden; when, however, he was drawn into more active fields during the war, the weakness again returned and has become more progressive. The condition of the bladder and his difficulty in swallowing have not changed since his first appearance.

Dr. Spiller thought that the condition was one of trophic disturbance, causing a true hypertrophy of the muscle groups. In his paper before the International Medical Congress (*Brain* **34**: Part 1, 1913), he accepted the classification of Batten on the myopathies, but added to it the true hypertrophic type. He reported another case of this disorder at that time.

For a time, true muscular hypertrophy was thought to be due to venous thrombosis, for it was frequently observed following typhoid fever, and the enlargement usually came on quite rapidly. The generalized muscular hypertrophy in this case, as well as in the case of Léri, which showed extensive muscle group involvement following encephalitis, puts venous thrombosis out of the question.

It is remarkable that in so many of the cases reported by Krabbe (*Revue Neurologique*, No. 7-8, 1921) there was a previous history of typhoid fever, about a year before the hypertrophy developed. After careful study, he could find only twenty cases in the literature. Two patients suffered from paralytic conditions from which they recovered. In most of the cases, only one or two limbs were affected. There is only one other case cited by him in which all the extremities were involved. No cases were mentioned in which the hypertrophy was generalized throughout the trunk and back, as in the patient here presented.

Babinski compares the condition to hypertrophic ostitis. In regeneration, overgrowth of tissue is likely to occur, as in keloids, hypertrophic ostitis and amputation neuromas; and, physiologically, in the case of the heart and uterus. In true muscular hypertrophy, the regeneration following multiple neuritis leads to overgrowth of the muscle. This view has also been taken by Léri and Krabbe.

Weitz has reported two additional cases (*Deutsch. Ztschr. f. Nervenh.* **71**: No. 4-6, 1921) and his theory is that adult organs cease to grow because of the firmness of the structures, but that in disease there is a loss of the restraining tissue firmness that holds the muscle tissues to their normal size, and, with the weakening of this barrier, there develops hypertrophy of the fibers and consequent enlargement of the muscle.

However, with the present evidence at hand, it would seem that the condition has followed certain diseases that are capable of producing a severe neuritis, and the attendant muscle pains and cramps would bear this out. It is, therefore, possible that true muscular hypertrophy may be due to some form of trophic irritation which has persisted, following a multiple neuritis, possibly of toxic or infectious origin.

SPONTANEOUS BRAIN LESIONS OF MONKEYS. Dr. Baldwin Lucke.

In order to study the spontaneous lesions in the nervous system of the primates, the brains from twenty-two monkeys and apes which had died at the Philadelphia Zoological Garden were examined. It was found that nine monkeys had lesions similar to those found in human pathology. Of particular interest was the occurrence, in three animals, of a nonsuppurative meningo-encephalitis, resembling the lesions found in human epidemic (lethargic) encephalitis, acute poliomyelitis and certain other diseases. Cerebral arteriosclerosis occurred in four animals, in two of which marked calcification of the vessels and focal areas of necrosis and gliosis were present. In one animal, a solitary tubercle was found in the pons. In two animals, acute suppurative meningo-encephalitis was present. This was associated in one case with a pontile abscess. The conclusion may be reached that the interpretation of experimental lesions in monkeys requires great caution, since definite brain changes were found in more than 40 per cent. of the animals studied.

DISCUSSION

Dr. Williams B. Cadwalader: In considering the question of diseases of the nervous system in wild animals kept in captivity, one should never lose sight of the fact that these animals are existing under abnormal conditions. They are received after being transported from different parts of the world under most unfavorable conditions. The monkeys and apes on arrival are placed in quarantine, and are subjected to tuberculin tests. Only nontuberculous animals are exhibited. What effect tuberculin tests may have on the nervous system is not known. The diet of each animal is carefully regulated, but, in spite of this, gastro-enteritis is the most common cause of death; though monkeys and other apes are subject to respiratory diseases. The exact ages of the monkeys on arrival cannot be determined, but they are not usually adults. The chimpanzee in which Dr. Lucke found alterations of the blood vessels of the brain died of influenza, but, though carefully examined before death, had shown no cerebral symptoms. In a large series of specimens now in the laboratory of the Gardens, there are few brains that show any primary disease. This was reported by me to the Pathological Society in 1912, and at that meeting the statement was made that arteriosclerosis rarely occurred. It seems probable that the changes Dr. Lucke found have some relation to respiratory infections or toxemias from gastro-intestinal disease. Furthermore, the brains Dr. Lucke examined are old ones that have been preserved in formaldehyd solution for a long time. What effect this may have on the staining reactions of the tissues is uncertain. Speaking generally, it has been our experience at the Philadelphia Zoological Gardens that primary disease of the central nervous system in animals and birds rarely occurs.

Dr. C. M. Byrnes: It is remarkable that arterial sclerosis should be so pronounced in a monkey 11 months of age. What is the average duration of life for apes?

Dr. Baldwin Lucke: The duration of life of apes varies. It averages about five years, in captivity. This study illustrates the necessity for further investigations of spontaneous lesions in laboratory animals, for it is difficult to evaluate experimental lesions while our knowledge of spontaneous tissue changes is incomplete. All this applies, of course, particularly to animals in captivity, which are more likely to be used for laboratory studies than those in their natural state.

DISEASE OF THE OTOLITH APPARATUS, WITH REPORT OF A CASE. Dr. Robert J. Hunter.

My case shows a pathologic condition in which, I believe, the apparatus governing static equilibrium is involved, while that governing dynamic equilibrium remains intact.

Recently, Magnus and Kleijn rotated animals in a centrifuge at two thousand revolutions per minute, thus dislocating the otoliths in the otolith membranes. By examining the reflexes of the animals before and after rotation, and by examining the otolith organs histologically, they have come to the conclusion that it is the otolith organs exclusively that are concerned in sending out these tonic labyrinth reflexes to the body muscles, and in the maintenance of the position reflex and compensatory eye movements. They conclude that the otolith apparatus is not stimulated by centrifugal force or, in the case of rectilinear motion, by any lagging of the otoliths on progression; and they call attention to the fact that, in the case of the canals, the stimulus is on movement either starting or stopping, whereas, in the otolith membranes, the stimulus is constant from the time the head moves from the horizontal position. These investigators believe that, even in this position, there is a constant minimum stimulus for tonus. If this is the case, then disease of the otolith organs would cause signs induced by the position of the head rather than by rotation of the head.

Some interesting work on the dog-fish, in which the otoliths or the ampullae can be destroyed without injury to other parts, has been done by S. S. Maxwell. He concludes that most of the reflexes are activated by both semicircular canals and utricle, and finds the reflexes only destroyed when both organs are extirpated. He believes the saccule has nothing to do with equilibrium. This may be true of fishes, but not of the higher vertebrates. He also believes that the canals are not stimulated by the flow of endolymph, but by the pull of the utricle on the ampullae.

Bárány reported a case of disease of the otolith apparatus in the *Journal of Laryngology and Otology*, May, 1921. It is the only one I have been able to find.

My case differs from the one reported by Bárány, but is, I believe, due to injury of the otolith apparatus. C. C., a boy of 14 years was referred to me at the Lankenau Hospital. His history, with the exception of three falls, was negative. His mother noticed that he held his head sideways soon after he had the second fall. The patient presented as his most prominent physical sign a marked horizontal nystagmus with excursions varying greatly. His mother was of the opinion that the nystagmus grew worse when he became excited. The nystagmus was horizontal when the head was held with the horizontal semicircular canals parallel to the floor. In this position, with a pencil at the near point, the movement was so small and fine that it was difficult to describe its direction. On looking to the right, he had horizontal nystagmus to the right, which was quick and of small amplitude. On moving the eyes to the left, he had horizontal nystagmus to the left, the amplitude being greater but the frequency less. When he looked at a distant point, he had marked horizontal nystagmus which was very quick, of good amplitude, and with the quick component to the left; when he looked at an object close at hand, he had noted that the violent movement stopped. When he put his head to the left side at an angle of 15 degrees, the nystagmus stopped. On holding his head straight or looking at a distance, he grew dizzy and sick at the stomach. He defined dizziness as a state in which objects moved back and forth, the

room not going around. When he looked up, the nystagmus continued to be horizontal and to the left; when he looked down, it was very slight. With the eyes closed, and the head at 30 degrees forward, both eyes appeared to be turned inward and slightly upward. When the eyes were opened after forcible closure, they looked straight ahead, then turned upward and outward. With his hand 60 degrees back, he held his eyes still for some seconds, when they again oscillated. With his head over either to the left or to the right shoulder, he could almost hold the eyes still. With his head 30 degrees forward, he had spontaneous past pointing, when tested in the perpendicular plane, of 2 inches (5 cm.) to the left with the right arm, and 3 inches (7.5 cm.) to the right with the left arm. With the head in the same position, the past pointing in the horizontal plane gave 2 inches (5 cm.) below with the right arm, and touches with the left arm. With the head 60 degrees backward he touched normally when moving the arms in the perpendicular plane. With his right leg (when the head was 30 degrees forward), he spontaneously past pointed to the right. With the left leg, he touched normally. When the head was 60 degrees backward, he touched with both feet. With the head held in the patient's favorite position, he past pointed normally with his arms moving in the perpendicular plane, also with his arms moving in a horizontal plane. With the right leg, he was a little uncertain in pointing, going sometimes to the right and sometimes to the left, but with the left leg he touched.

Both ear drums were retracted and there was an old scar in the posterior quadrant on the right side. The hearing was normal. The caloric and rotation tests were normal. The eye and neurologic reports were negative.

Since the nystagmus became more marked on changing the position of the head and as the usual Bárány tests showed that impulses went normally through all pathways and the results of the tests were normal, I feel that this was probably a case of disease of the otolith apparatus in distinction to disease of the semicircular canals. I base this opinion on the fact that the canals were normal and the position of the head seemed to aggravate his symptoms. The patient carried his head to one side and was comfortable in that position, but when he held it upright, nystagmus became very marked, he became nauseated and dizzy, and the tests revealed abnormal conditions.

Book Reviews

AN ESSAY ON THE PHYSIOLOGY OF MIND. An Interpretation Based on Biological, Morphological, Physical and Chemical Considerations. By FRANCIS X. DERCUM, A.M., M.D., PH.D., Professor of Nervous and Mental Diseases in the Jefferson Medical College. Cloth. Price, $1.75 net. Pp. 150. Philadelphia: W. B. Saunders Company, 1922.

The modern critical or scientific study of Mind was inaugurated by the school of psychologists which dates from Bacon and Descartes, and has its greatest names in Locke, Berkeley, Hume and Kant. Its method was truly scientific in that it broke away from the old scholastic speculation, and relied on a critical study of mental phenomena. Hence the reproach which is sometimes brought against it, that it was metaphysical, is unfounded; and can only be raised by those who are dissatisfied with the study of consciousness as equivalent with the study of mind. The method of Locke, Berkeley and Hume was by introspection; they ignored the anatomy and physiology of the brain, of which indeed they knew little, although Locke had studied medicine and was in full accord with its spirit and aims. The problems they set before them were the nature of knowledge, sensations, ideas and mental processes in general; and they believed that these problems could be successfully approached by looking inward at the activities of the mind by the only avenue possible, namely, by consciousness. That this method has its limitations, no one will deny; but this fact does not detract from its merits. Indeed, it is a method which must always continue in use; to abandon it is inconceivable. The modern science of psychiatry depends largely on it, and always will depend on it. We study the abnormal processes of the mind by the methods of psychologic introspection; and the Freudians even tell us that by this method they can probe beneath the surface and reveal the secrets of the subconscious. The study of brain cells and neuroglia, of dendrites, lymphocytes and infections, may supplement this method, but it is safe to say that it will never supplant it.

This psychologic school threw no light on the problem of substance in its relationship to mind; they were not interested in morphology, because, apparently, they believed that we cannot know the nature of matter; therefore, that we cannot hope to know how consciousness is a phenomenon of matter. If any of their critics have solved this problem, they have, up to the time of this writing, kept the secret to themselves.

It was inevitable, however, with the growth of the sciences of histology and pathology, that the attempt should be made to correlate their results with the well-established principles of psychology. As physicians we know only too well that infections and the diseases of brain cells have their psychologic reactions. We also know that heredity and environment are controlling factors in the evolution of character; in the manifold phenomena of behavior. The risk has been, however, that mere formalism, or materialism, such as is acquired by the too exclusive use of the microscope, would obstruct a broad view of problems which are vital to the physician as well as to the psychologist. Many of these problems will never be solved in the laboratory.

Dr. Dercum has sought to give a biologic interpretation of "mind," and his book is useful as presenting this aspect of the subject. He has evidently wanted to avoid metaphysics, and calls attention to the fact that the word "psyche" has not been used by him. In order to make a good start he begins

properly with the lower forms of life, for it is evident that, from the biologic standpoint, the very beginning of psychic function is to be observed in these lower forms, as was maintained by Haeckel. The elementary biologic processes, such as in the selection and appropriation of food by the ameba, are doubtless biochemical or physicochemical in nature; yet they represent the earliest example of "choice," which has not yet risen to the level of a volitional act. It is a so-called "tropism"; and it is one of the earliest, if not the earliest, of the instincts. The physiology of the cell, as shown not only in the selection of food but also in the transmission of impulses and in motor reactions, as well as in a tendency to differentiation, or a division of function—all this physiology is discussed clearly by Dr. Dercum, and it is used by him as the basis of his thesis. If the reader keeps this in mind, the unfolding of the argument proceeds naturally and easily. The point of view is that of the biologist and evolutionist; and the great value of the book, and the satisfaction which arises from reading it, come from the fact that it is logical and well presented.

We cannot venture, in a brief review, to go into details. Dr. Dercum follows closely the evolutionary scheme, as it has been worked out through the ages, and presents step by step the unfolding of the cerebrospinal system. From the lower to higher levels, from spinal segments to the neopallium, from instincts to intelligence, the story of the neurons is told up to the supreme level of the mental faculties. When he reaches the stage of "consciousness" he is reticent, and perhaps wisely so, for no one has ever defined consciousness. We have to accept it as one of the ultimate facts. It defies definition. "Whatever it is," he says, "it is constantly changing." In this fact, perhaps, we see its elusive nature; but this does not absolve us from the necessity of studying it, as did the old psychologists, if we are ever to gain an adequate knowledge of the physiology of the mind.

Dr. Dercum, in discussing the physiology of the neuron, reverts to his theory of the retraction of the processes of the neuron as the cause of some of the symptoms of hysteria; the contacts of one cell with another are broken, hence occur the paralyses, anesthesias and various stigmas of the disease. This mechanistic theory is brought forward by the author very appropriately to illustrate the biologic conception of his book. The attempt to give a purely physiologic explanation of such higher mental faculties as will, initiative, thought, etc., is also made, but the subject is one of extraordinary difficulty, and it is perhaps too soon yet to attempt it. But the explanation of the physiology of the unconscious field, that is, that it is merely a reawakening of memories by the power of association, is a valid one, and it can readily be assimilated to the "laws of the association of ideas" which were established by the old psychologists. The psychanalyzers would do well sometimes to take heed of this. It remains for them to prove, on biologic grounds, that there are any active processes in the subconscious, such as they describe, or that it is anything other than a storehouse of memories.

It is very interesting to note that, although Dr. Dercum disclaims being a metaphysician, he takes a position on the subject of human knowledge that is closely akin to that of Bishop Berkeley; for he seems to hold (p. 119) that "we can only be conscious of the changes in the protoplasm of our own substance." In other words, we only know our own mental processes, and have no certain knowledge that these processes represent an external reality. This comes very near to Berkeleyan "idealism"; and shows in an interesting way that the great problems are the same after all, whether they are approached on the one side by the "psychologist," or on the other side by the "biologist."

HUMAN BEHAVIOR. In Relation to the Study of Educational, Social and Ethical Problems. By STEWART PATON, M.D. New York: Charles Scribner's Sons, 1921.

The preface to this book must serve as the preface to the review. "The importance of the subject is obvious and has been tragically emphasized by the present world crisis. Little is known about man as he is. Imagination has supplied many of the details in the picture of what he was once supposed to be, while disappointment associated with unrealized expectations of what he might have become has increased the difficulties of taking measures of his present stature. Parent, teacher, physician, student of social phenomena, prospective reformer, statesman, and philosopher, each has his special interest in the great human problem. Today every intelligent citizen is anxiously awaiting the solution to the problem of how 'democracy may be made safe for the world.' There can be little doubt that in the careful, painstaking study of man as he is will be found the means by which human institutions may be established upon a more rational basis and at least an intelligent effort made to lay the foundations of a durable peace."

Readers of the ARCHIVES will be interested in the neuropsychiatric aspects of any study of "human behavior." Neurologists will find most familiar ground in the account of special mechanisms of adjustment which makes Chapter 3 a very generalized account of the central nervous system. Psychiatrists will find Dr. Campbell's chart of the levels at which mental disease may arise at the beginning of Chapter 2 and later in the same chapter and in Chapter 11 specific references to the unconscious in mental disease. Dementia praecox is mentioned only once. And yet the methods proposed for the betterment of a poorly managed world are psychiatric, and the psychiatrist will find much encouragement in noting the suggestions which are made about the social value of by-products of his own proper job.

Early in the book we meet these stimulating statements, which are here loosely quoted. Human beings are governed more by their emotions and less by their intellects than they think they are. Physical characteristics have more to do with the development of character than is recognized. Moralists have forced us this way and that without considering whether the later state was worse than the first; performing operations which were sometimes "pronounced a success, in spite of the intellectual and moral death of the patient." Man has been too proud to study his own behavior as he studies that of lower forms; he has created "an imaginary world, inhabited only by people at the prime of their activities" and ruled by a conscious reason which sharply differentiated them from the animal (and the child and the insane). Note the standards of the people of "Main Street." "Systems of law, ethics, religion, and education were deliberately planned with reference only to the needs of the relatively few persons who were capable of attaining a high degree of intellectual activity and volitional control."

If these things are so, then the establishment of human life on a better basis depends on the acquisition of self-knowledge. The moralist must study behavior in animals and man without reference to causes and dispassionately. The biologic reactions of man "may be governed by laws no harder to understand than is the law of gravitation." We must study them in the dog, ape, child, and in the insane because there are found to be the simpler reactions.

Finely spun definitions, such as efforts to define the conscious, are not going to be of great value. Into the response which the total individual as a unit makes goes something that he knows about and something that he knows

less about—called indifferently the unconscious, or habit, or organic memory or subconscious. There is a sliding partition between conscious and unconscious, and between voluntary and involuntary, and we can let it go at that.

The trouble with the central part of the book is that in its study of the organization of the personality, one misses the person. The synthesis which is promised is not there. The triad of autonomic nervous system, internal secretions, and the conditional reflex play a large part in explaining the development of the personality. The over or under function of any part disturbs the reactions tending to preserve the unity of the organism. So when "psychologic tension" is lowered it carries down with it the "quantity of action," the inventive power and the general "adaptive capacity," and we have a quiet depression. But if the psychologic force is not lowered in proportion to the lowering of the tension an agitated depression results. Many figures of speech occur which might lead the reader too far; why should the stream of energy, if dammed from its appropriate channels, displace the affect? We look at this and that piece of mechanism, but do not see its integration or its therapeutic helpfulness. We are thrown back for helpful suggestions on the value of instilling good habits, of protecting children from emotional shocks, of studying all the motor impulses of children, of grading occupation and environment to objectively determined mental states. If all men could be studied and managed as are the favored insane!

The book has a good big idea and many valuable suggestions.

THE PRACTICAL MEDICINE SERIES. Comprising Eight Volumes on the Year's Progress in Medicine and Surgery. Under the General Editorial Charge of CHARLES L. MIX, A.M., M.D., Professor of Physical Diagnosis in the Northwestern University Medical School. Volume VIII. Nervous and Mental Diseases. Edited by PETER BASSOE, M.D., Associate Professor of Nervous and Mental Diseases, Rush Medical College. Price, $1.75. Pp. 249. Chicago: The Year Book Publishers, 1921.

The present volume maintains the high standard of previous years and is as good a summary of the year's output of neuropsychiatric literature as could be arranged in a volume of this size. The entire field is covered, with special attention to some subjects. For instance, there are twenty-nine pages on the various phases of lethargic encephalitis and several pages on related conditions. Seventeen pages are devoted to neurosyphilis.

Among the interesting things discussed, we may mention brain changes in poisoning by illuminating gas, colloid and calcareous degeneration of cerebral vessels, the parkinsonian syndromes, an excellent report on myelomalacia, injection of air into the spinal canal for diagnostic purposes and nervous symptoms due to vertebral anomalies.

There are sixty pages of excellent matter on mental diseases. We believe nothing of importance has escaped the eye of the editor. The illustrations are good and the index adequate.

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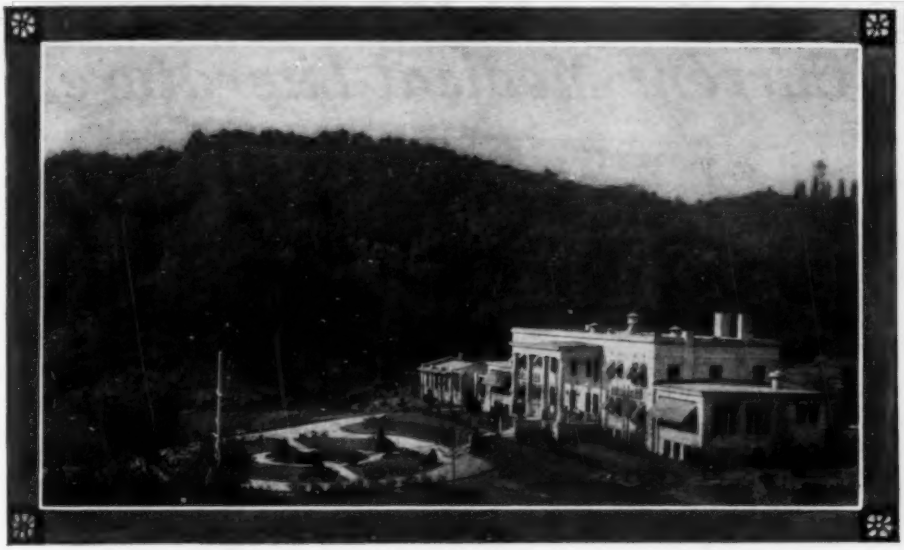

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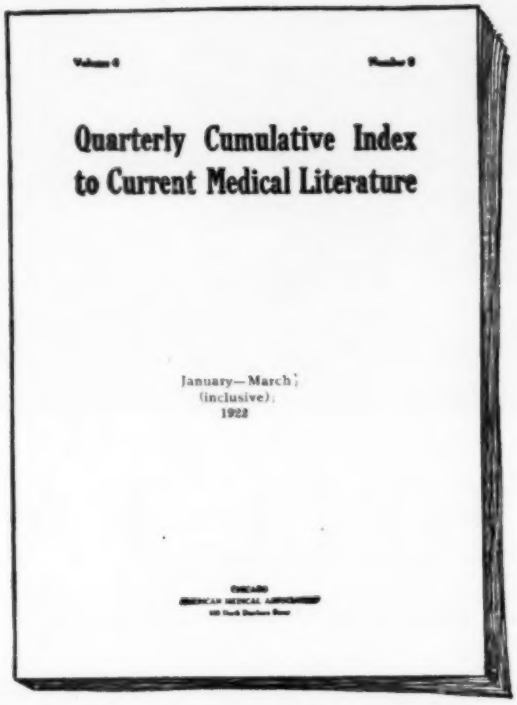

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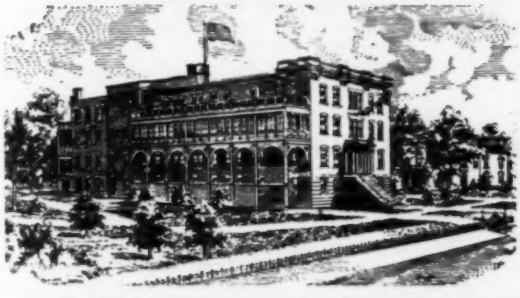

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